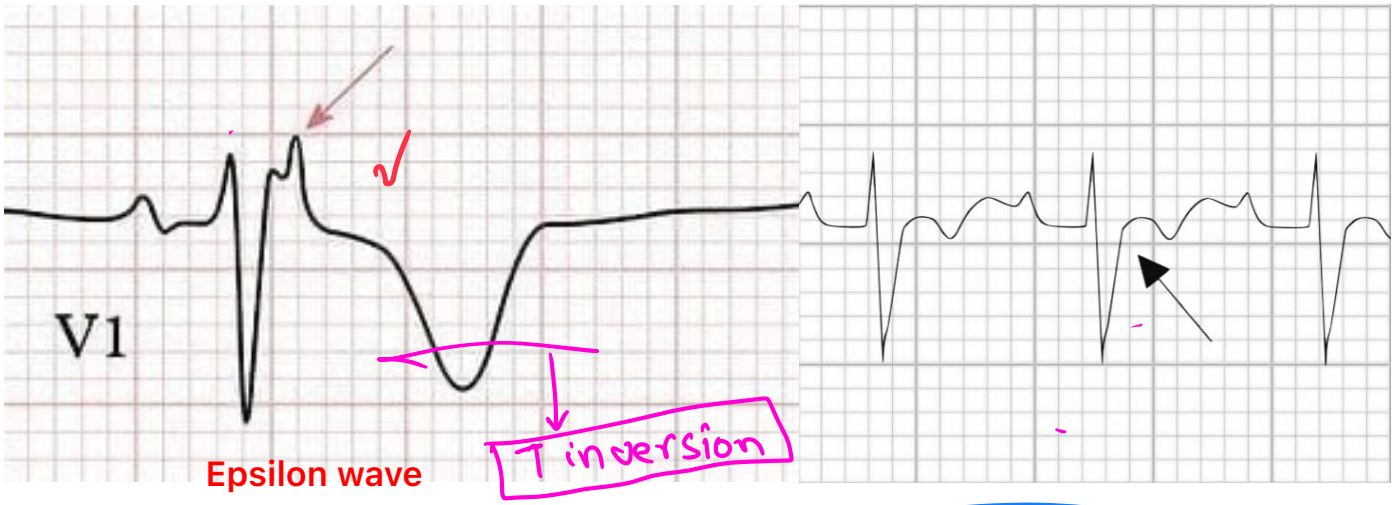
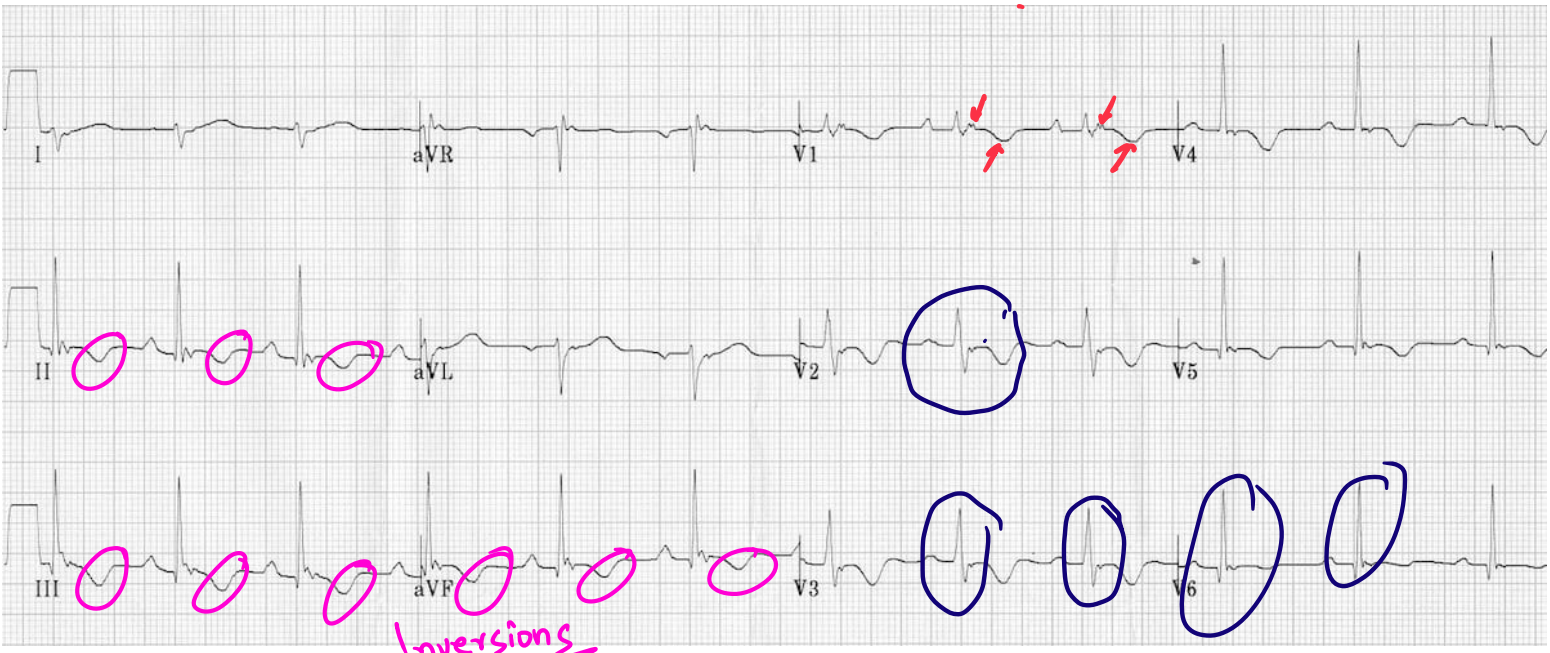
# Arrhythmogenic Right ventriculardysplasia




Prolonged S-wave upstroke in V2 with localized QRS widening

T wave inversion in right precordial leads V1-3, in absence of RBBB (85% of patients)
Epsilon wave (most specific finding, seen in 50% of patients)
Localised QRS widening in V1-3 (≥ 110ms)
Prolonged *S wave upstroke* of 55ms in V1-3
Ventricular ectopy of LBBB morphology, with frequent PVCs > 1000 per 24 hours
Paroxysmal episodes of ventricular tachycardia (VT) with LBBB morphology (RVOT tachycardia)

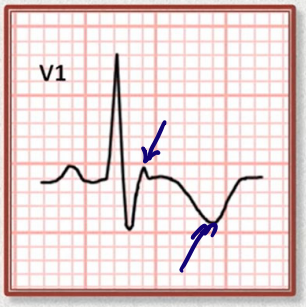
40 x 3 = 120
120 small boxes,




T-wave inversion in precordial and inferior leads, without RBBB pattern
Epsilon wave in V1
Localised widening of QRS in V1-2

# Arrhythmogenic Right Ventricular Dysplasia (ARVD)

## EKG Characteristics




**Epsilon Wave**

*Highly Specific*

*Sensitivity ~30%*

**Other manifestations include:**

- QRS duration > 110ms in V1-V3
- T wave inversions in V1-V3
- RBBB (complete or incomplete)
- Prolonged S wave upstroke ≥ 55ms

***However, the EKG at presentation may be completely normal.***

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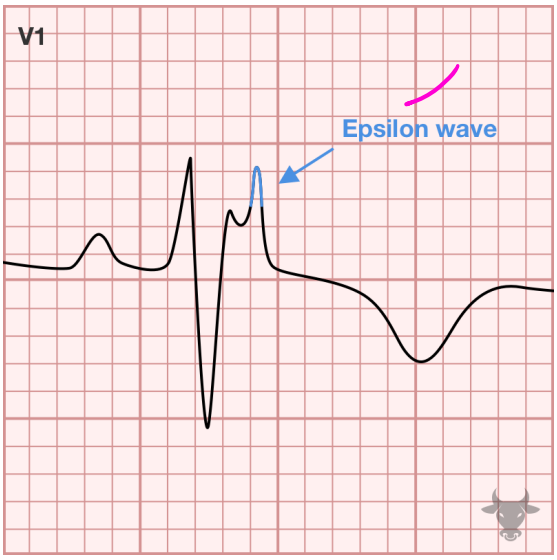
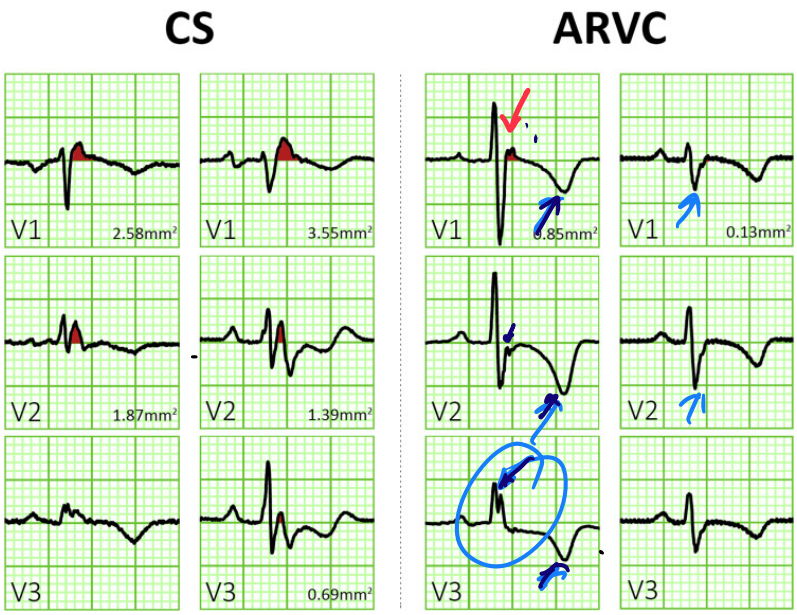
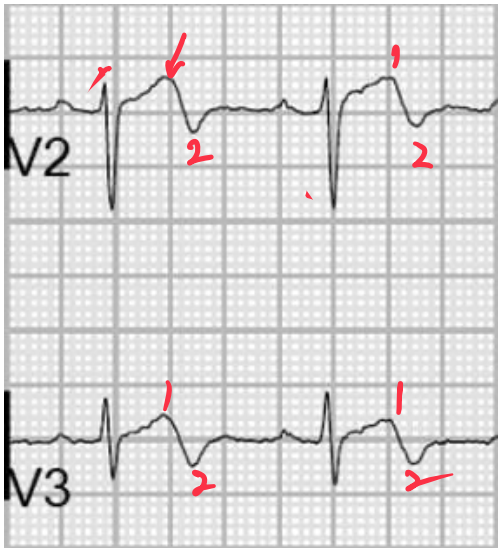


# Wellen's syndrome

**biphasic or deeply inverted T waves in V2-3**, plus a history of **recent chest pain now resolved**. It is highly specific for **critical stenosis of the left anterior descending artery (LAD)**



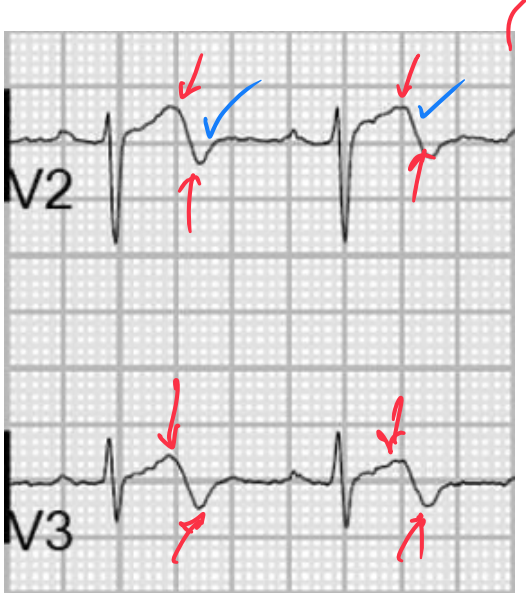

**Wellens pattern A:**
**Biphasic T waves**

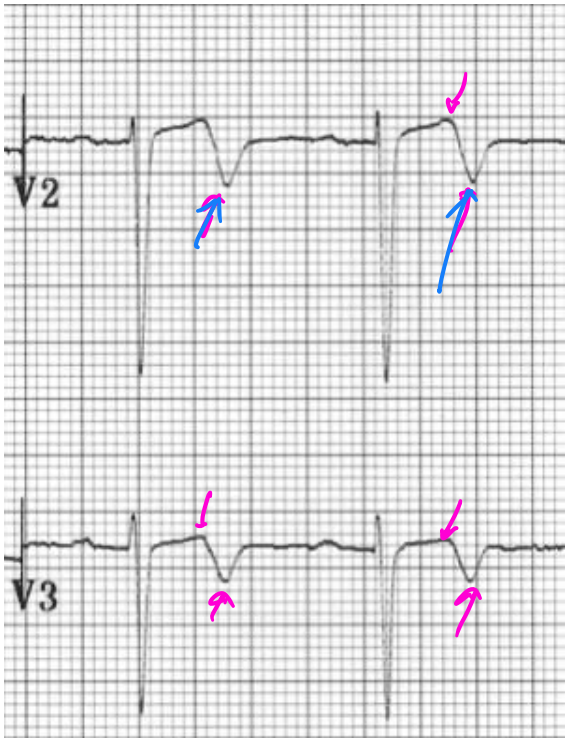
**Wellens pattern B:**
**Deeply inverted T waves**

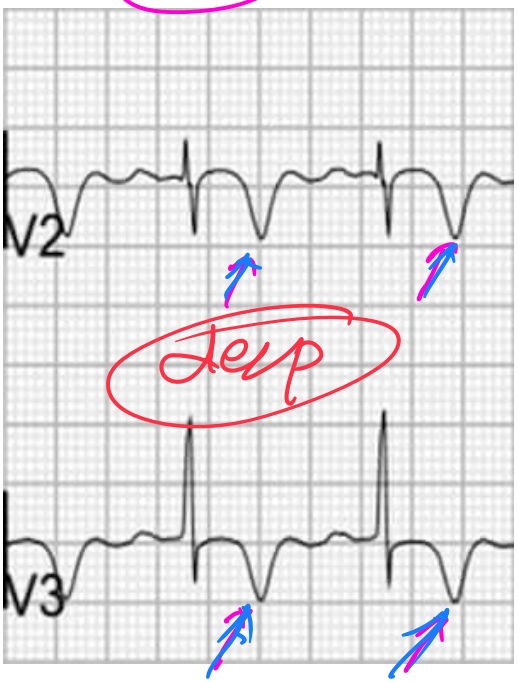
The pattern is usually **present in the pain free state** — it may be **obscured during episodes of ischaemic chest pain**, when there is "pseudonormalisation" of T waves in V2-3
Wellens syndrome is a key example of why all patients presenting with chest pain must have serial ECGs

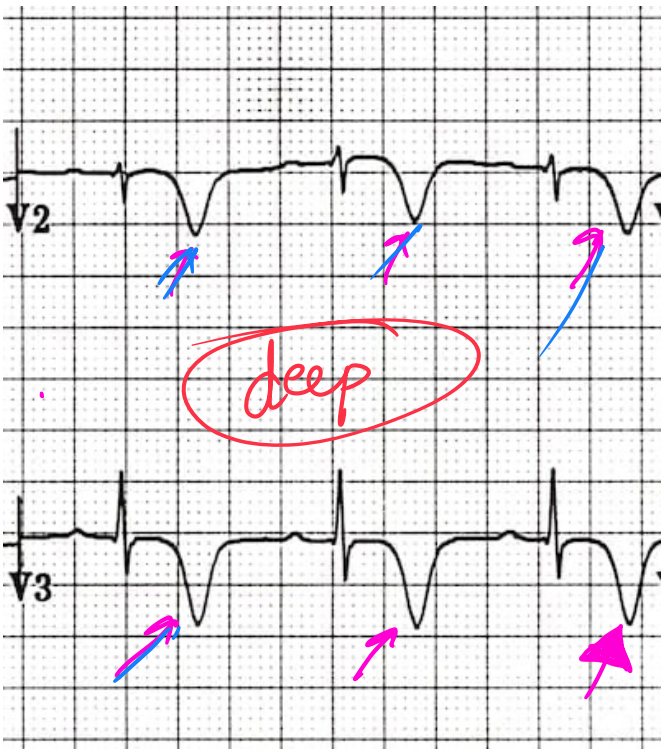
**extremely high risk for extensive anterior wall MI within the subsequent days toweeks**

Type (1)



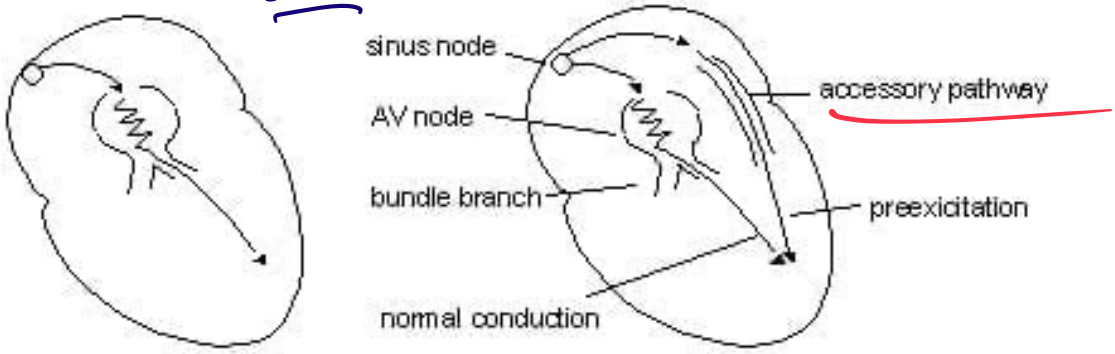



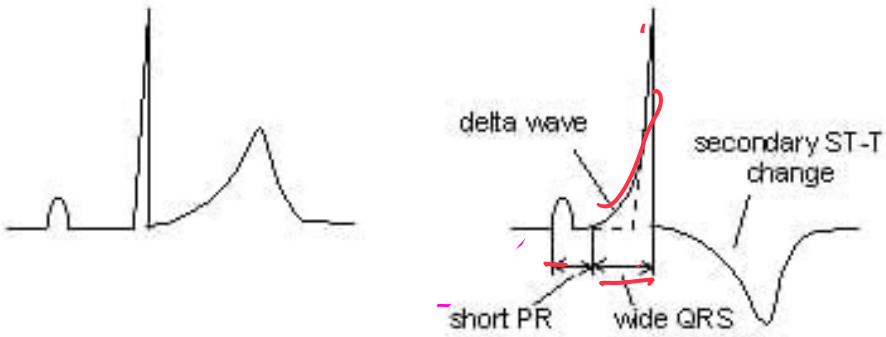



## Wolff-Parkinson-White (WPW) Syndrome

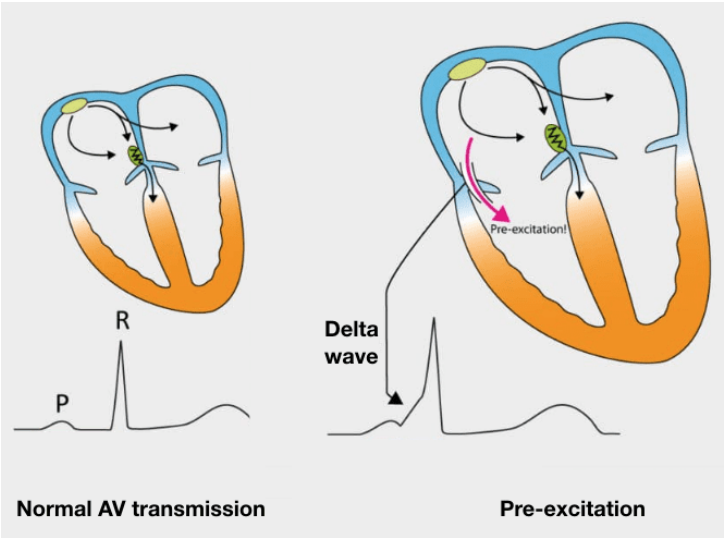
PR interval < 120ms
Delta wave: slurring slow rise of initial portion of the QRS
QRS prolongation > 110ms

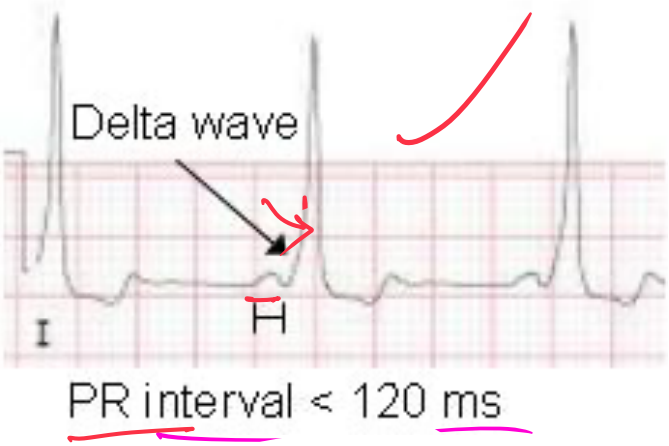


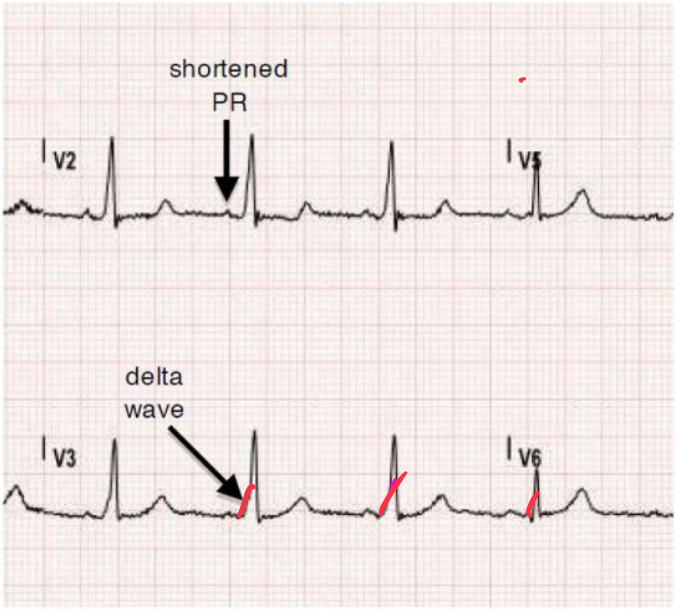



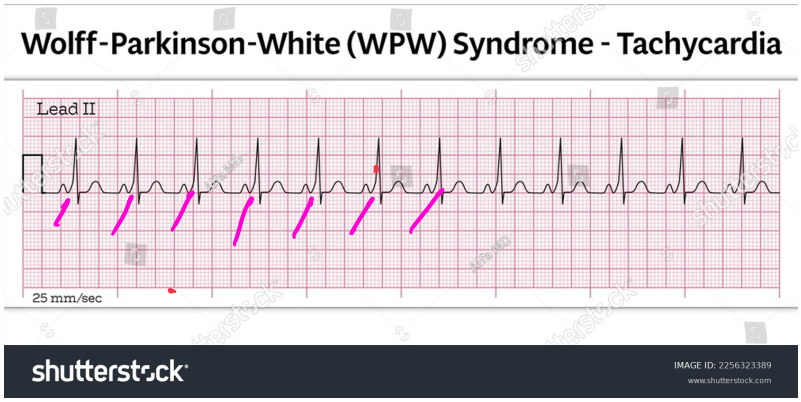

Normal conduction

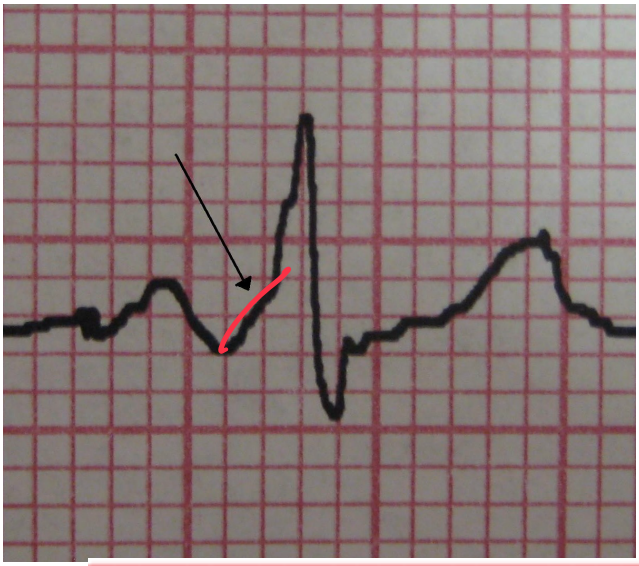
Preexcitation

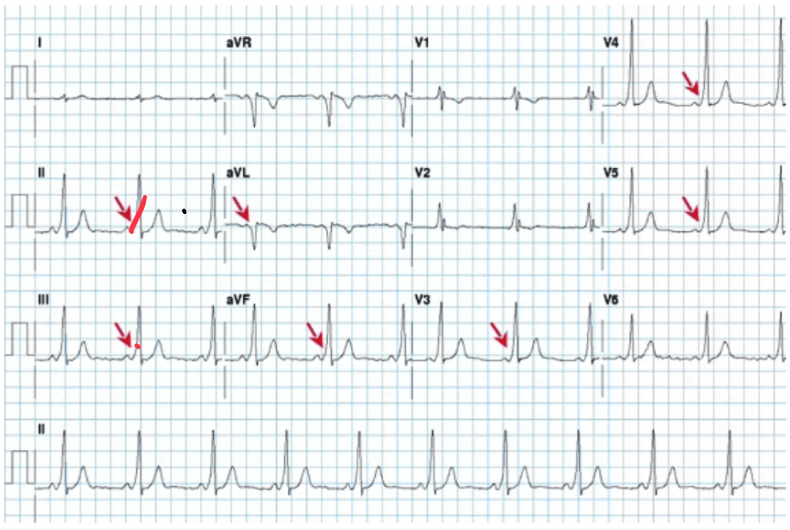

R
P
Delta
wave
Pre-excitation!
Normal AV transmission
Pre-excitation

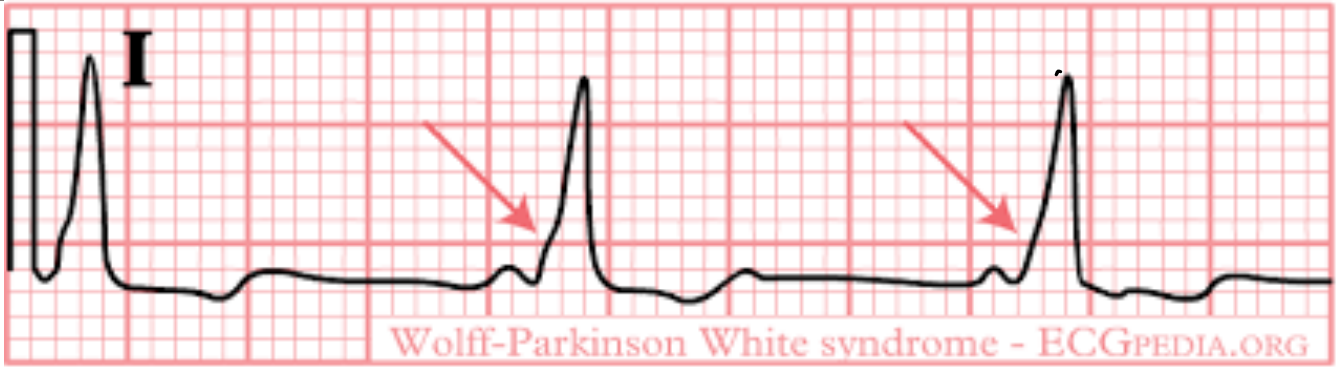

Delta wave
I
PR interval < 120 ms

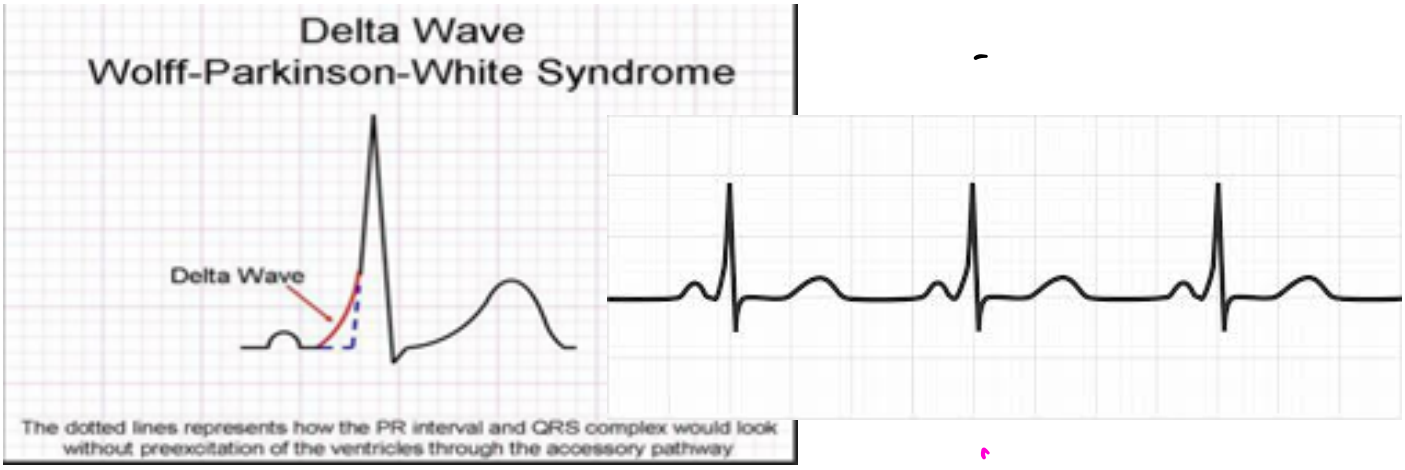

shortened
PR
V2
V5
delta
wave
V3
V6


Wolff-Parkinson-White (WPW) Syndrome - Tachycardia
Lead II
25 mm/sec
shutterstock
IMAGE ID: 2256323389
www.shutterstock.com

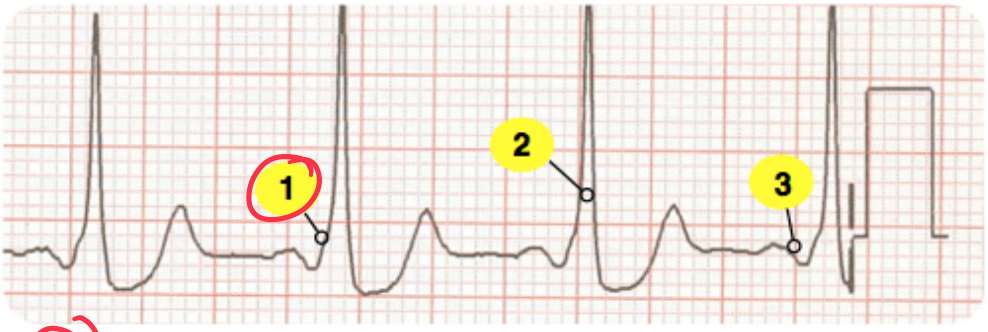



I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II


I
Wolff-Parkinson White syndrome - ECGpedia.org

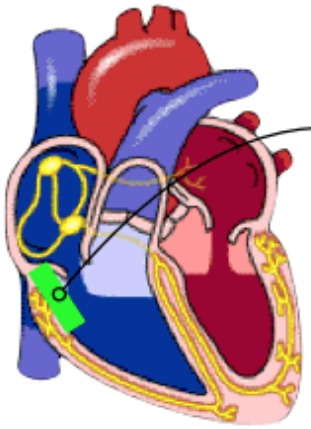



# Wolff-Parkinson-White Syndrome

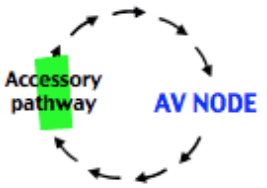



1. Slurred upstroke of QRS complex (Delta wave)
2. Wide QRS complex (QRS > 120 msec)
3. Short PR interval (PR < 120 msec)

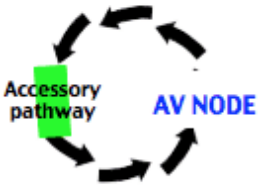
Triad



**Bypass tract (Accessory pathway)**
*Bundle of Kent*



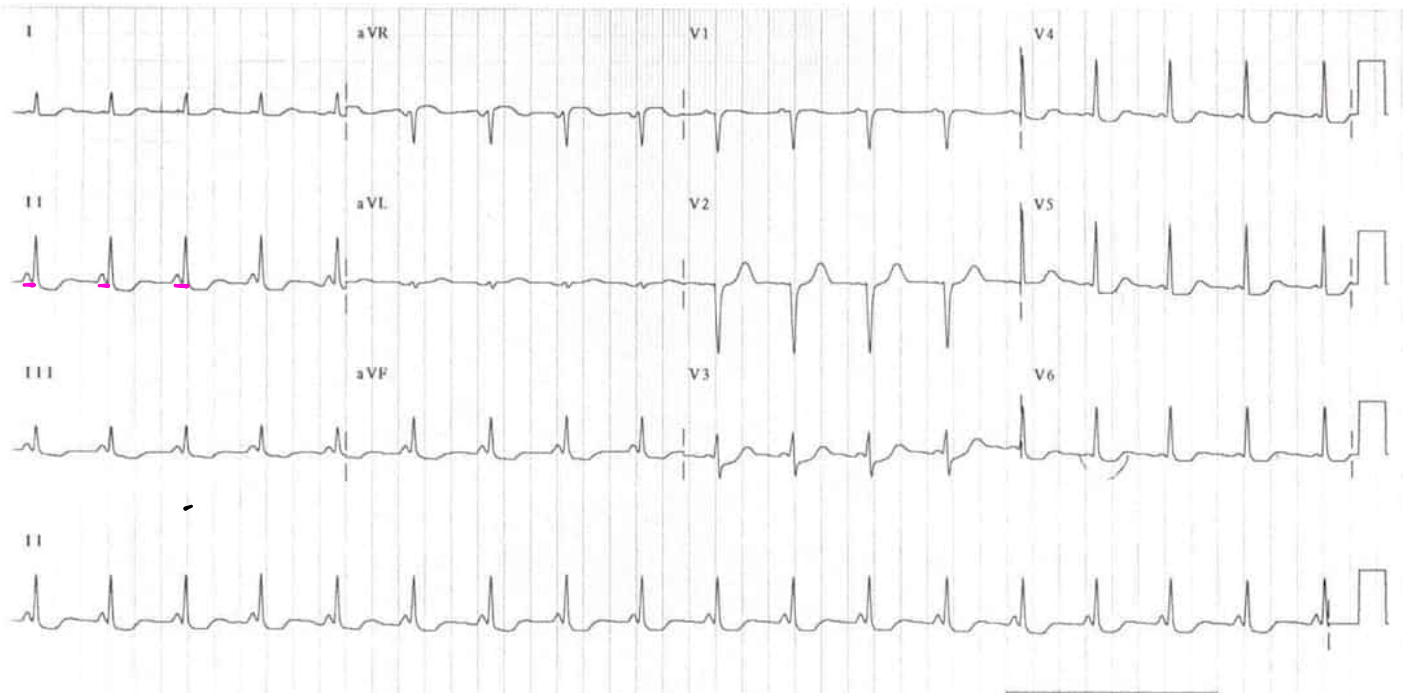

**Orthodromic**




**Antidromic**

## Lown–Ganong–Levine syndrome

Short PR interval (<120ms)
normal QRS complex
Absence of a delta wave

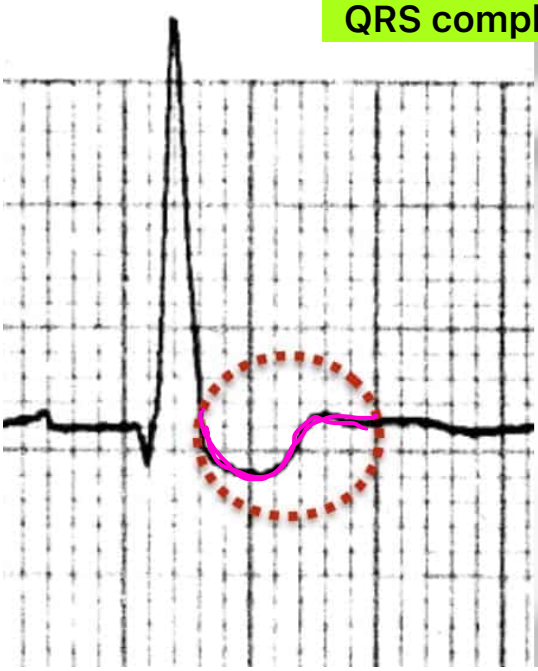


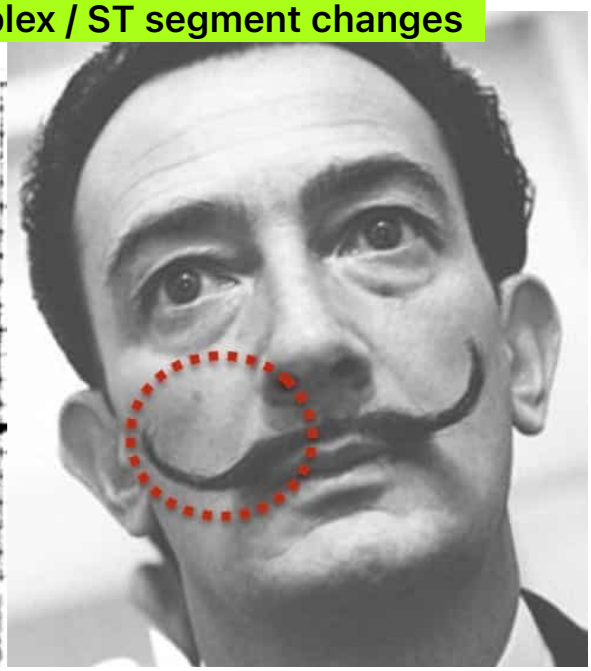

## Digoxin

Prolonged PR & short QT

- Down-sloping ST depression ('reverse tick', 'scooped out')
- Flattened / inverted T waves
- Arrhythmias e.g. AV block, bradycardia
- DIGOXIN DOESN'T CAUSE TYPE II 2nd degree HEART BLOCK

QRS complex / ST segment changes

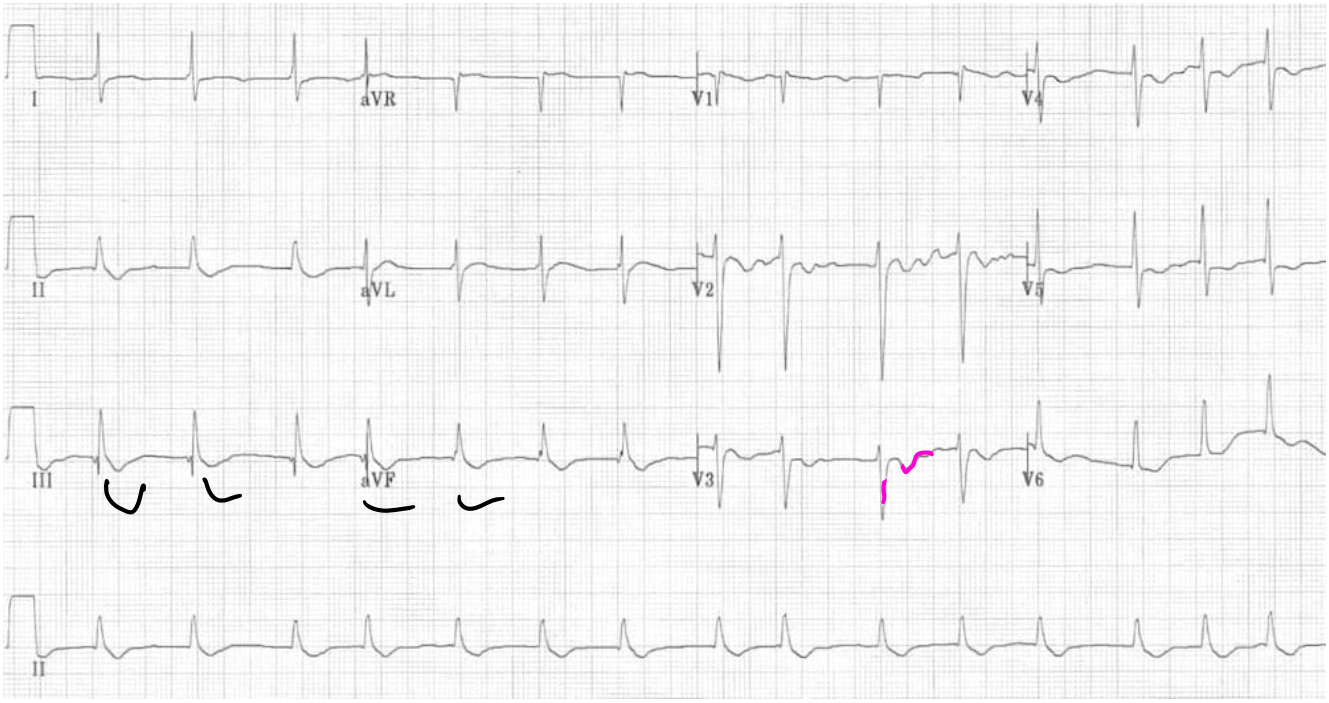




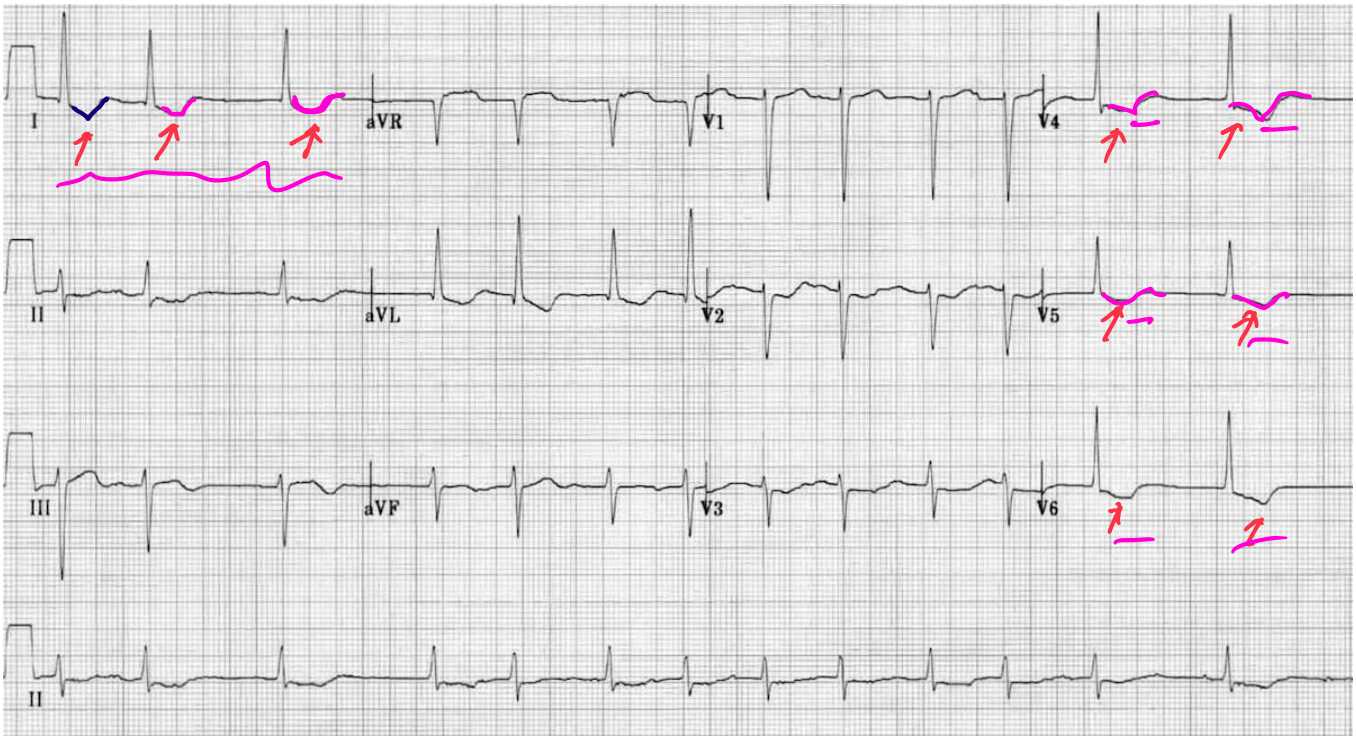
slurred", "sagging" or "scooped" and resembling either a "reverse tick", "hockey stick" morphology of QRS ST

T-wave changes

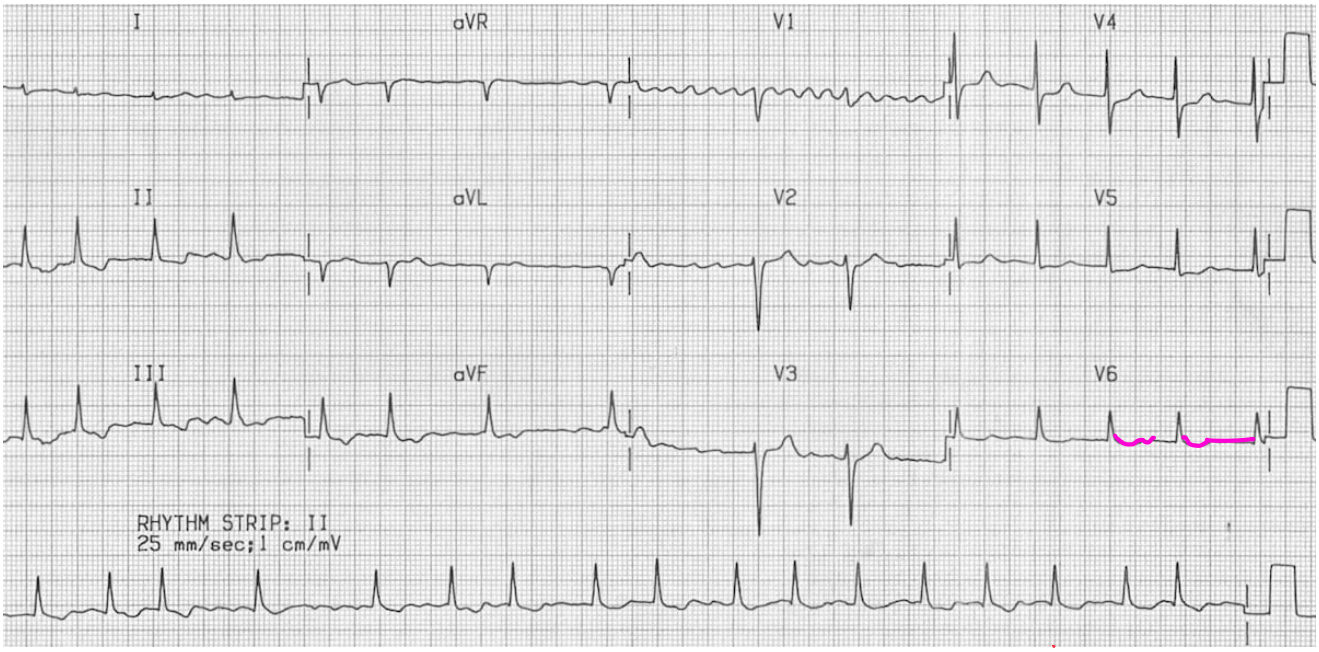
The most common T-wave abnormality is a biphasic T wave with an initial negative deflection and terminal positive deflection. This is usually seen in leads with a dominant R wave (e.g. V4-6).



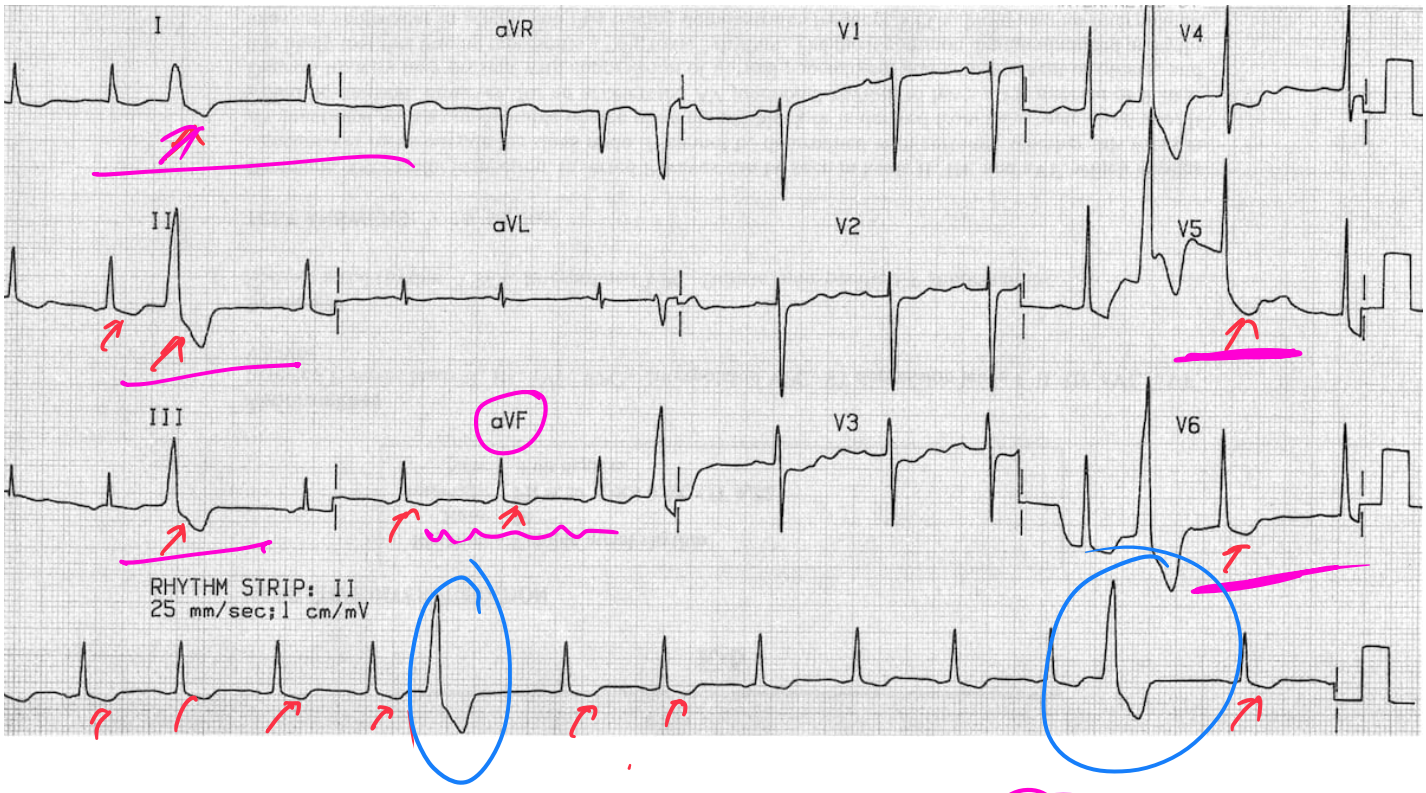

This is the classic picture of digoxin effect, with "sagging" ST segments and T waves taking on the appearance of "Salvador Dali's moustache"




Sagging ST segments are most evident in the lateral leads V4-6, I and aVL




The sagging morphology is most evident in V6 and in the lead II rhythm strip



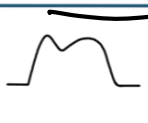

Sagging ST depression is clearly evident in leads I, II, III, aVF and V5-6
Frequent premature ventricular complexes (PVCs) suggest the possibility of digoxin toxicity

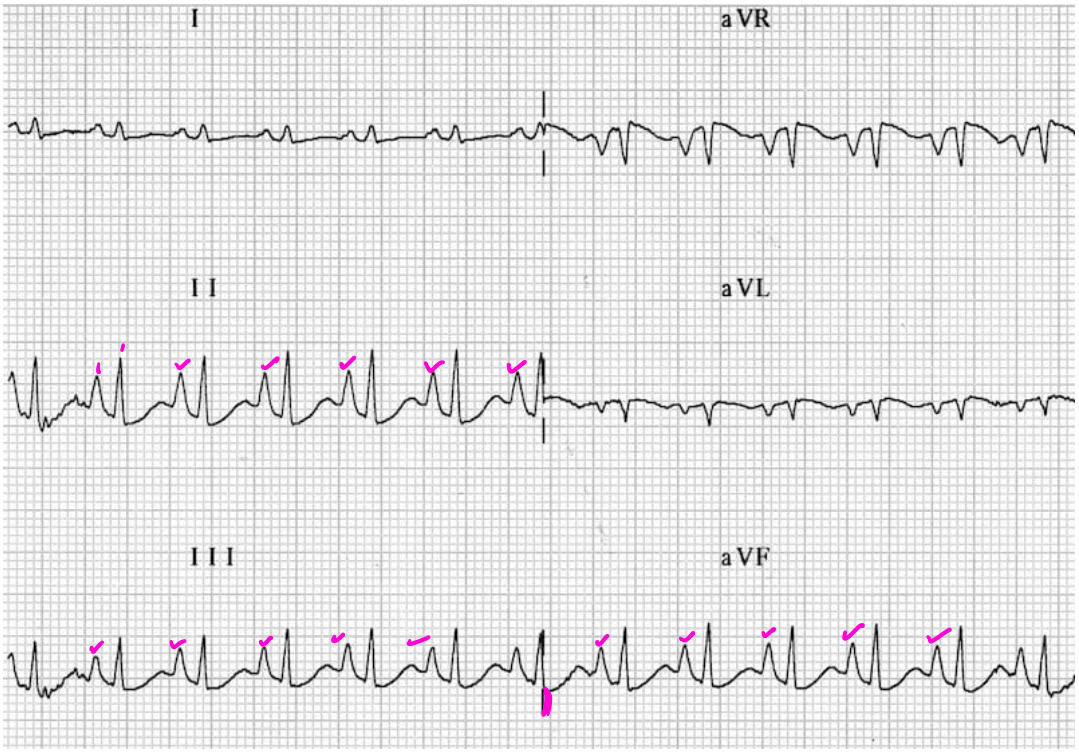
## Right atrial enlargement produces a peaked P wave (P pulmonale) with amplitude:

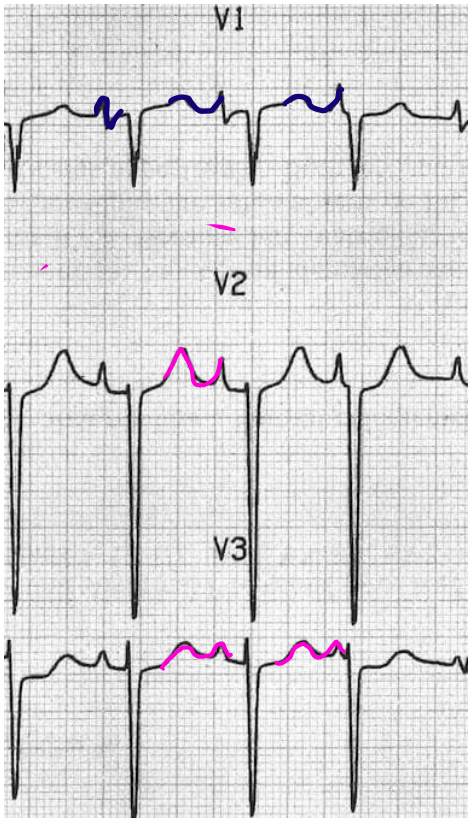
### Right Atrial Enlargement (RAE), Right atrial hypertrophy (RAH)

> 2.5 mm in the inferior leads (II, III and AVF) = 2.5 small boxes
> 1.5 mm in V1 and V2 → 1.5 small boxes

1 mm = 1 small box

| | II | V1 |
|---|---|---|
| Normal | | |
| RAE | | |
| LAE | | |
| RAE + LAE |  | |



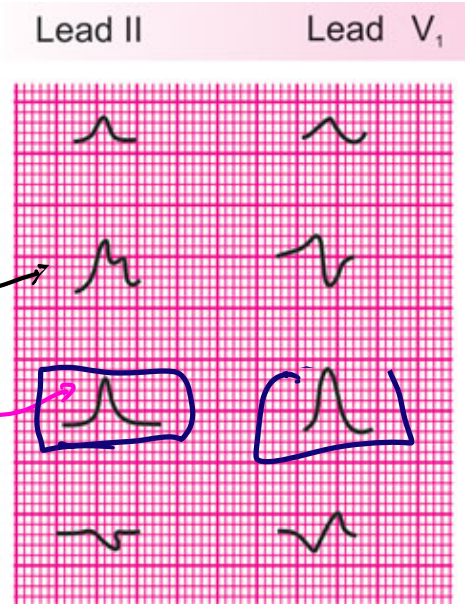



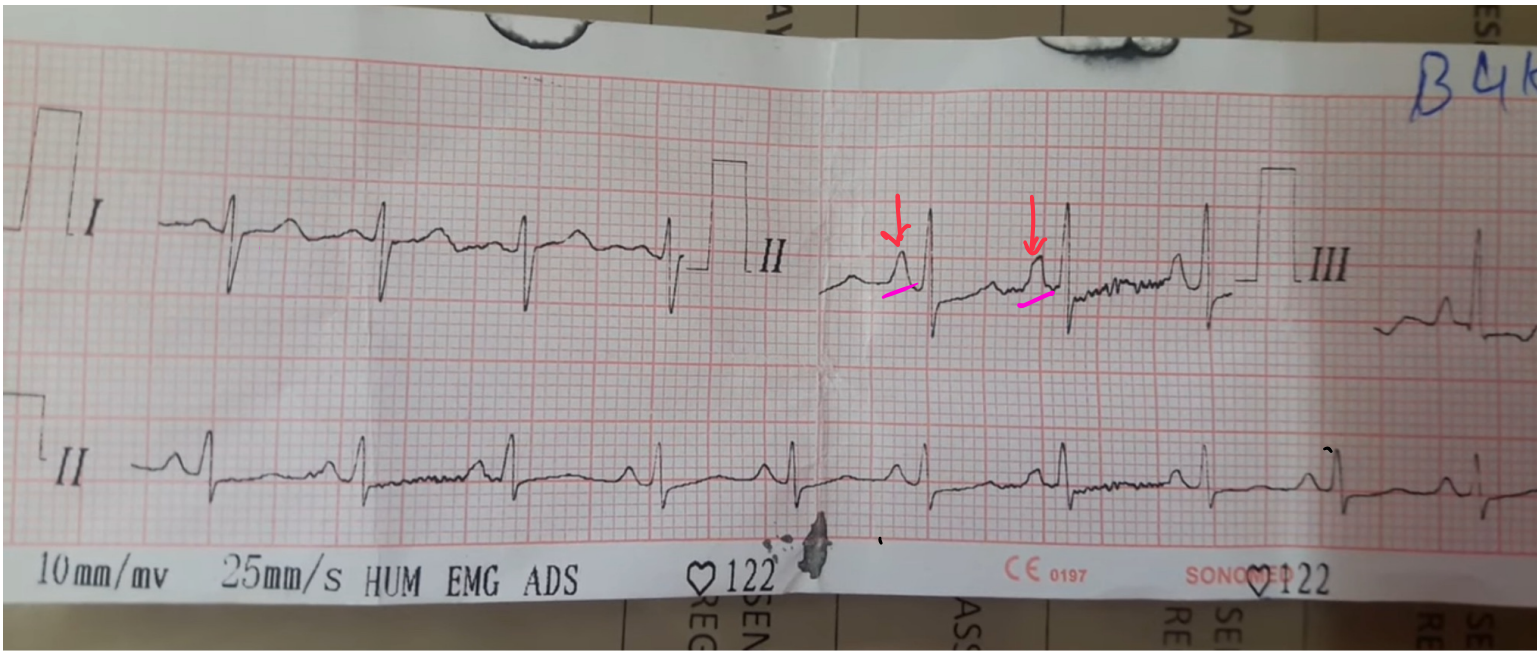

Normal P wave

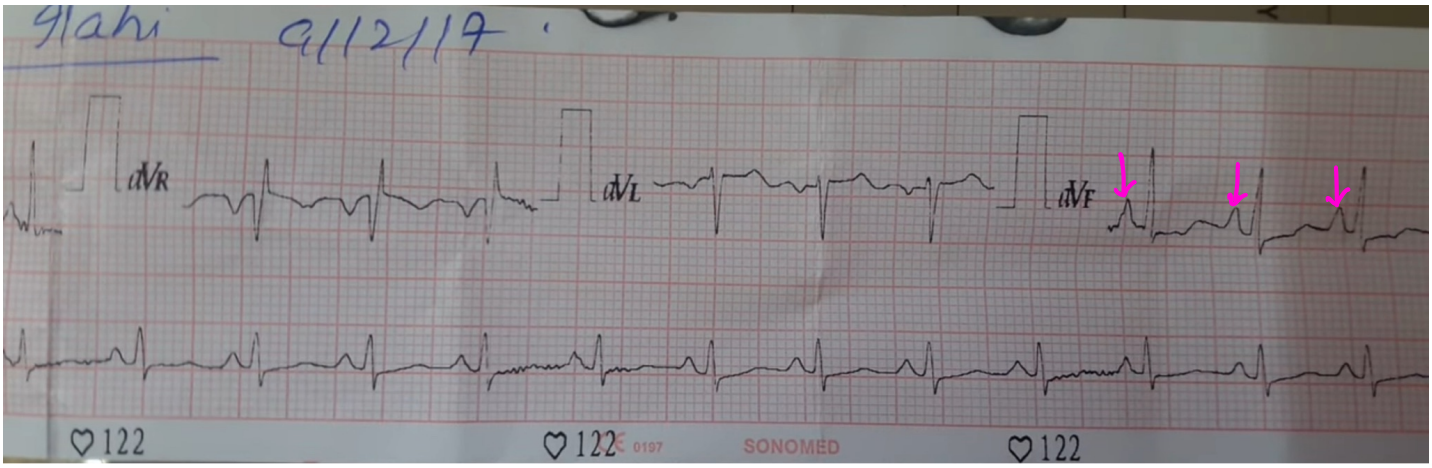
P mitrate
(left atrial hypertrophy)

P pulmonale
(right atrial hypertrophy)

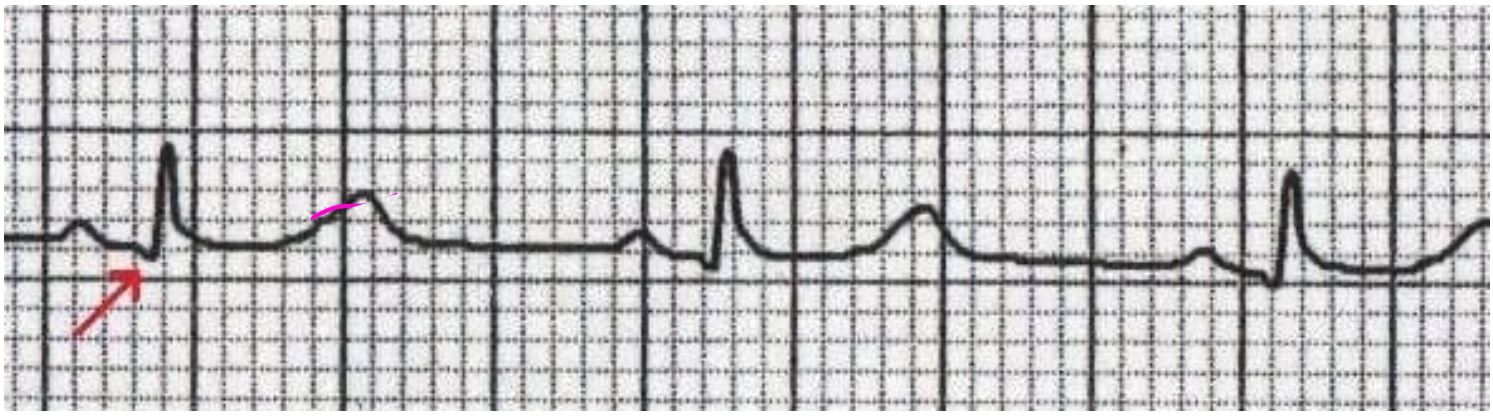
Retrograde P wave



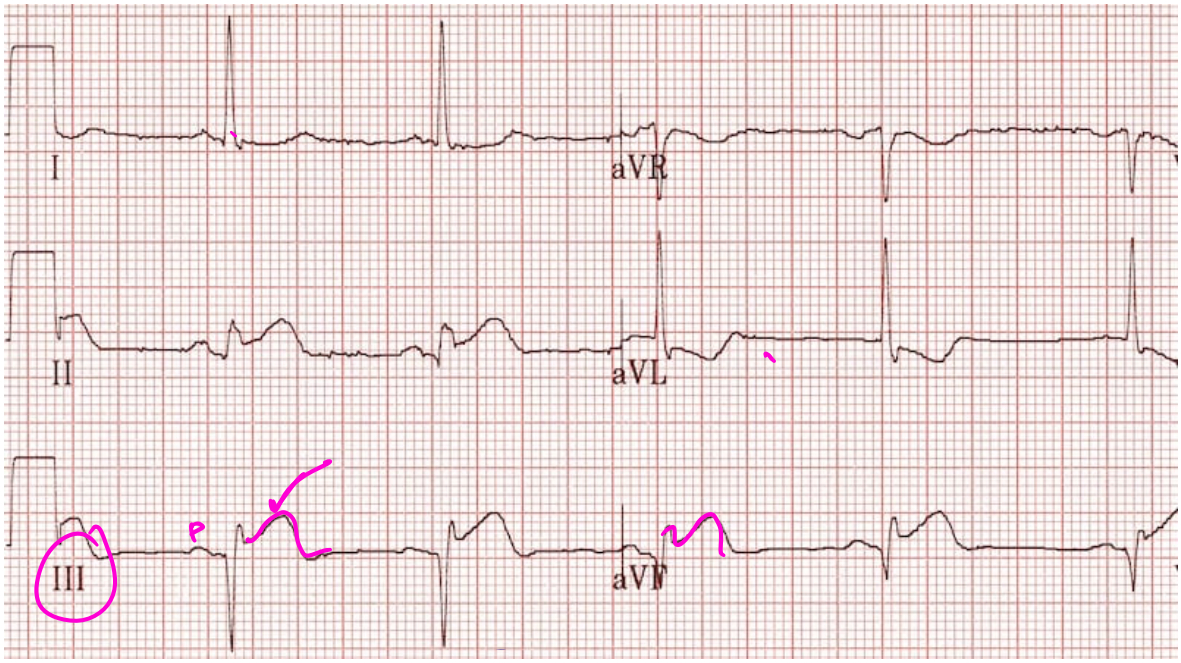


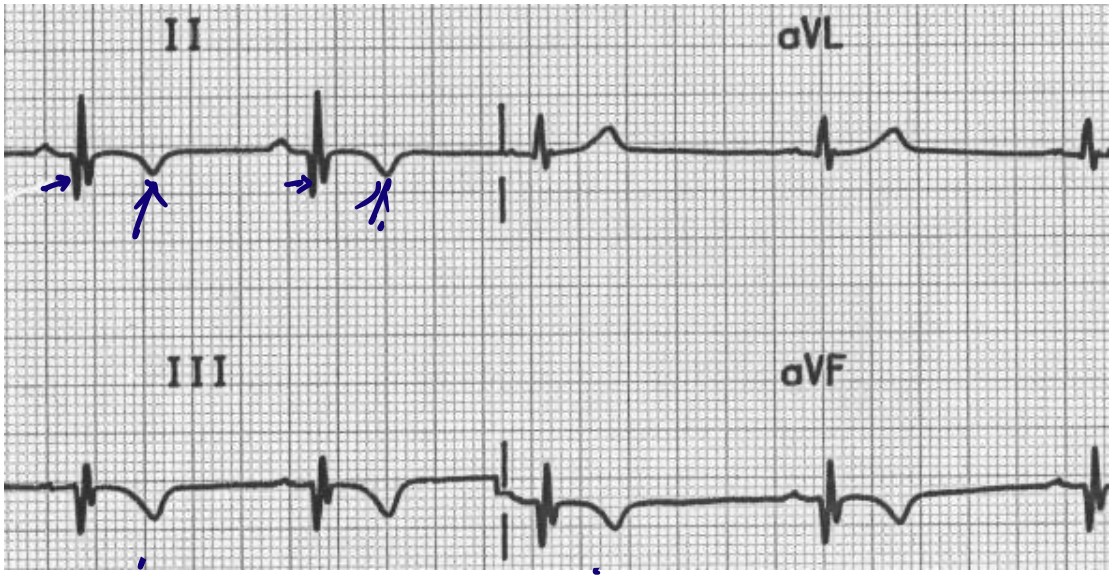

Q- wave → Normal



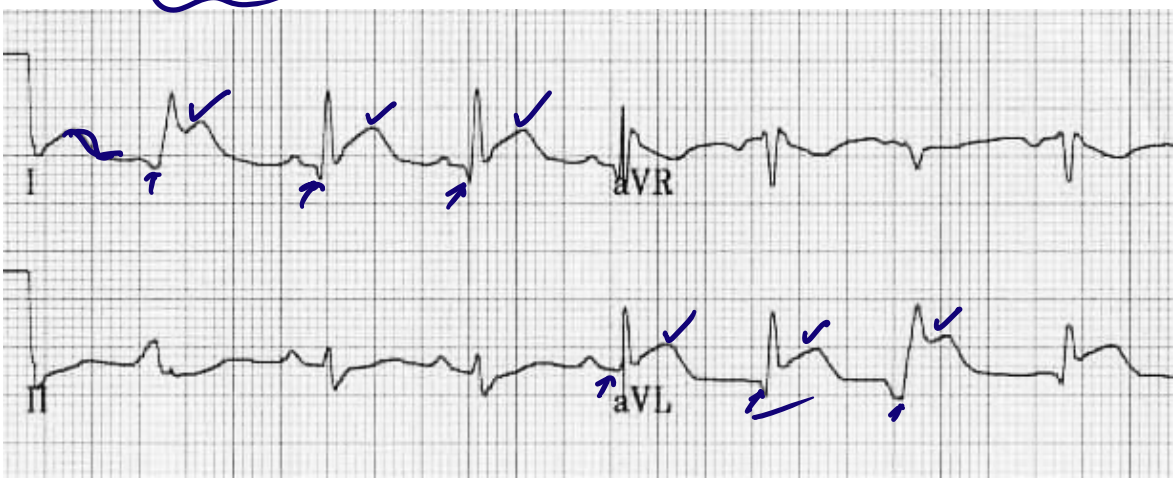
Q waves are considered pathological if:
> 40 ms (1 mm) wide
> 2 mm deep
> 25% of depth of QRS complex
Seen in leads V1-3



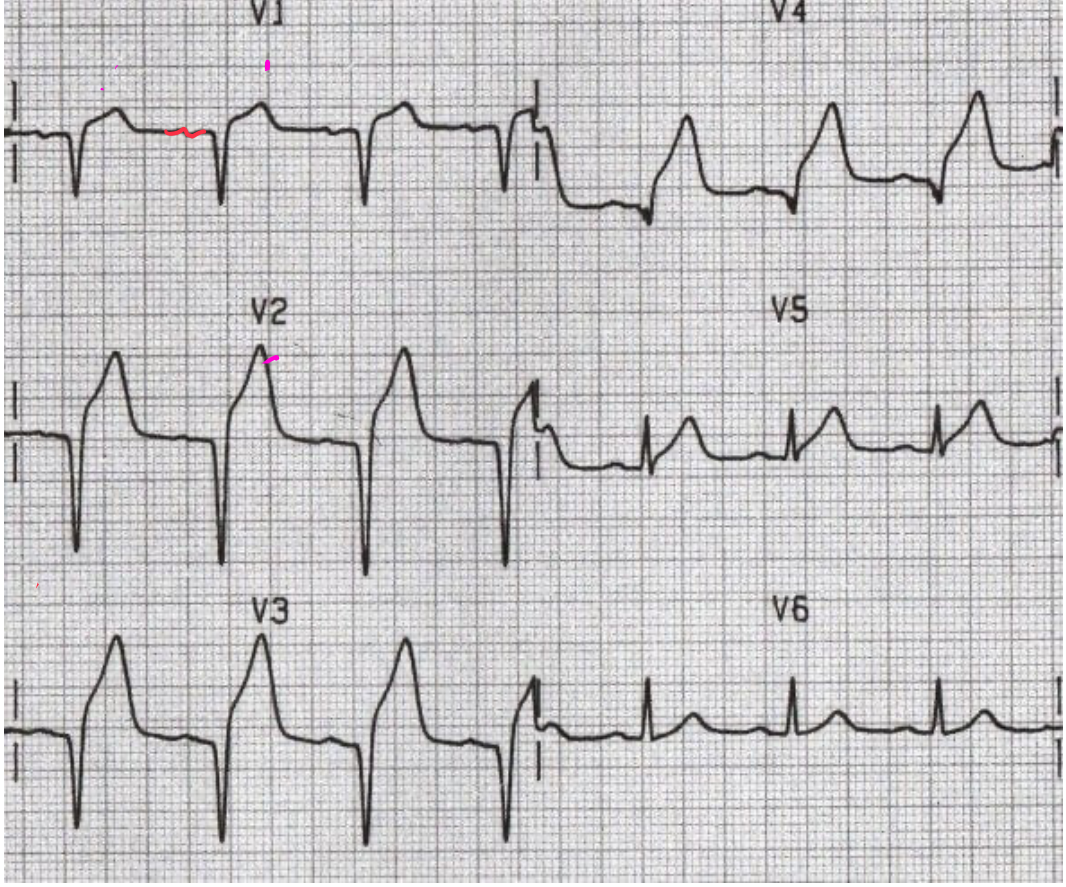

Inferior Q waves (II, III, aVF) with ST elevation due to acute MI



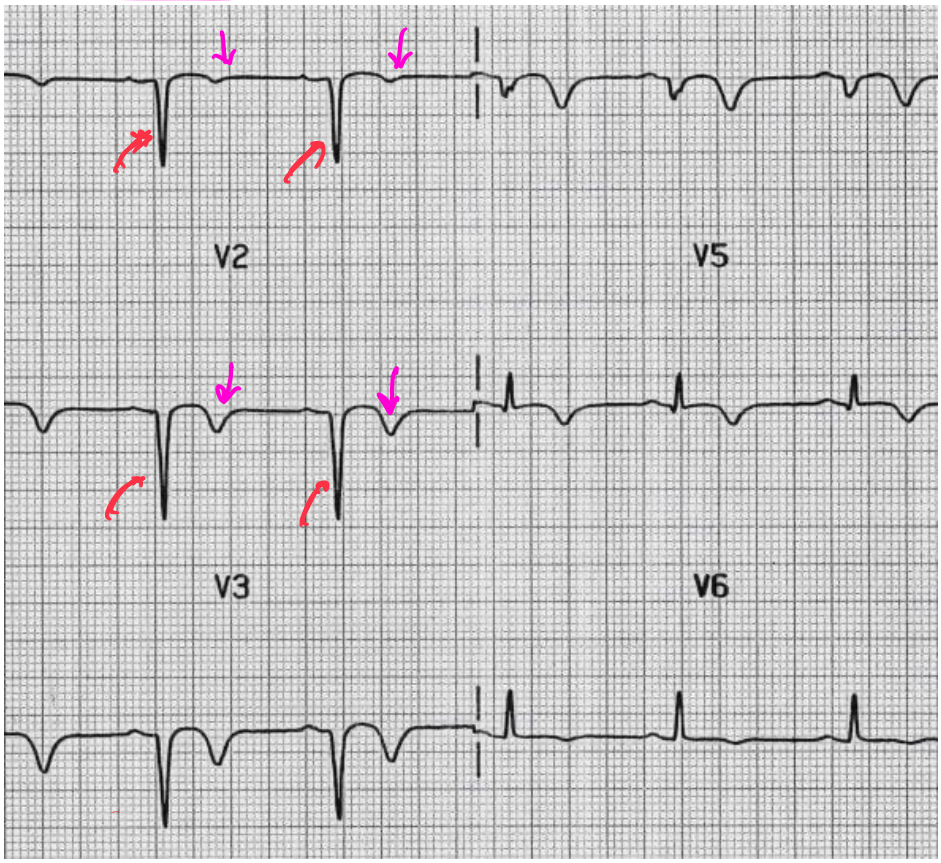

Inferior Q waves (II, III, aVF) with T-wave inversion due to previous MI




Lateral Q waves (I, aVL) with ST elevation due to acute MI

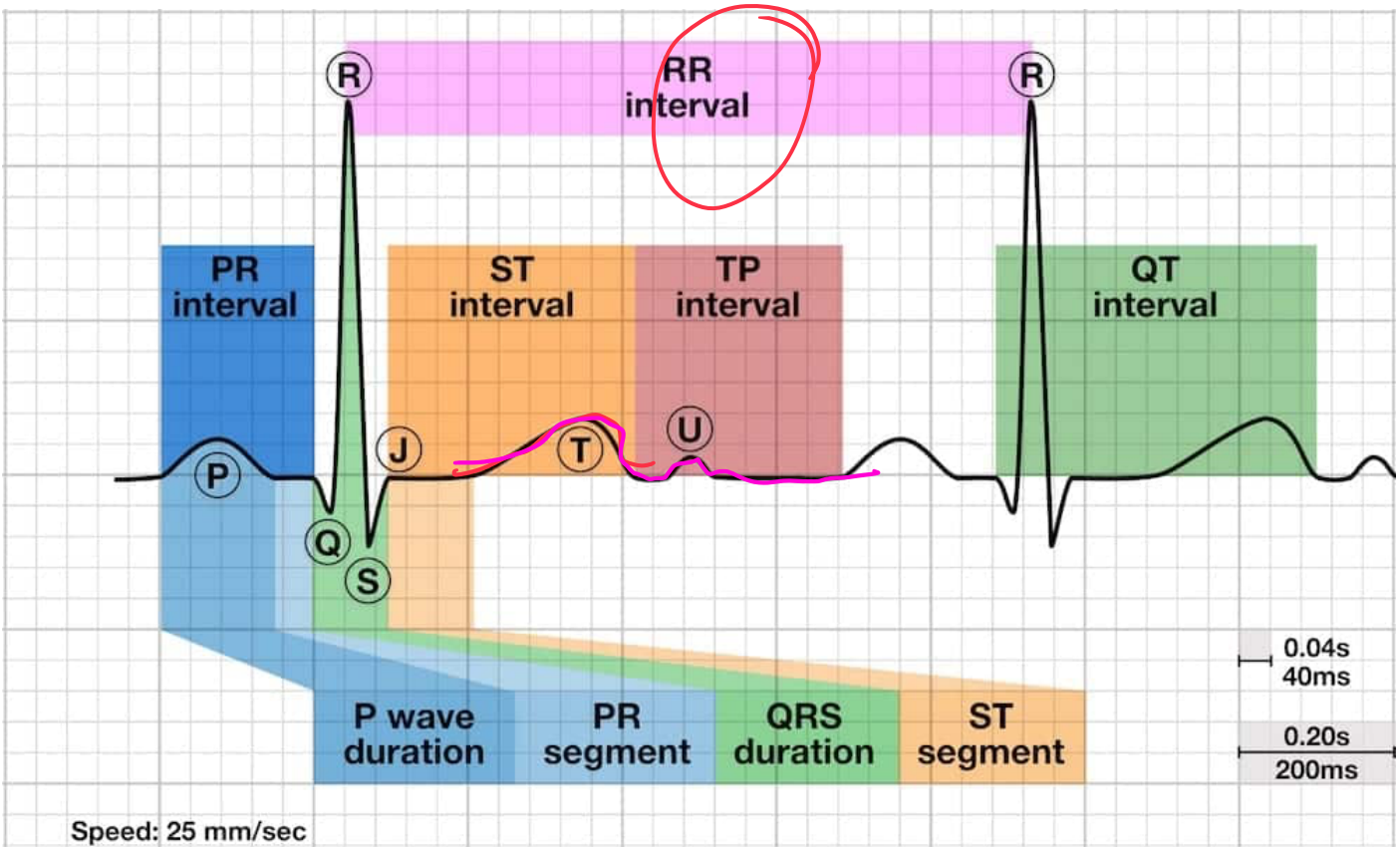


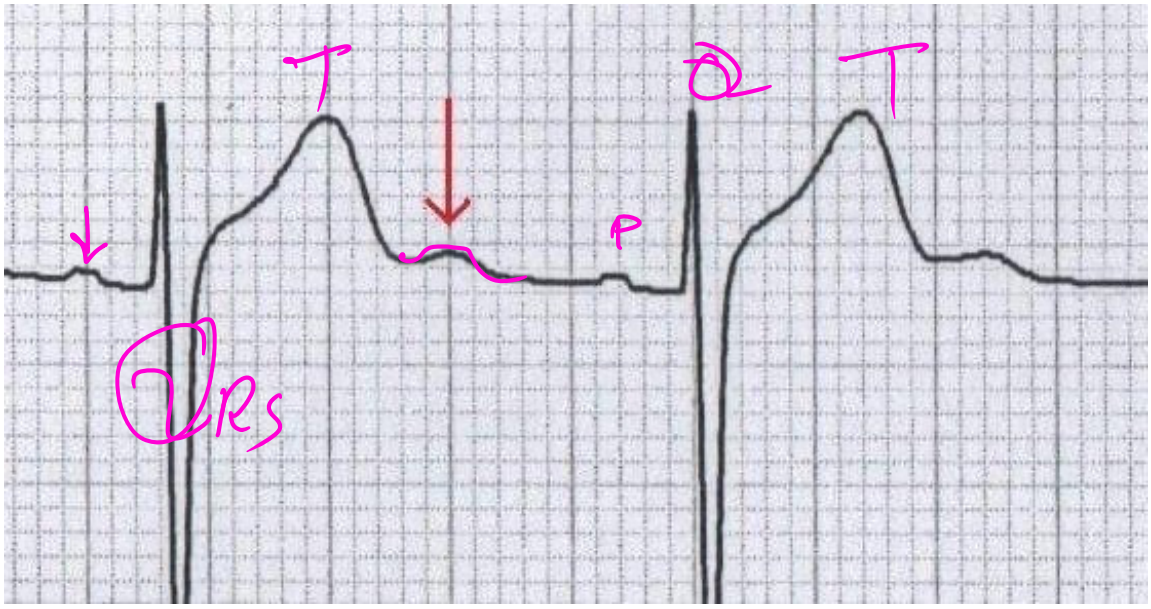

Anterior Q waves (V1-4) with ST elevation due to acute MI



The U wave is a small (0.5 mm) deflection immediately following the T wave

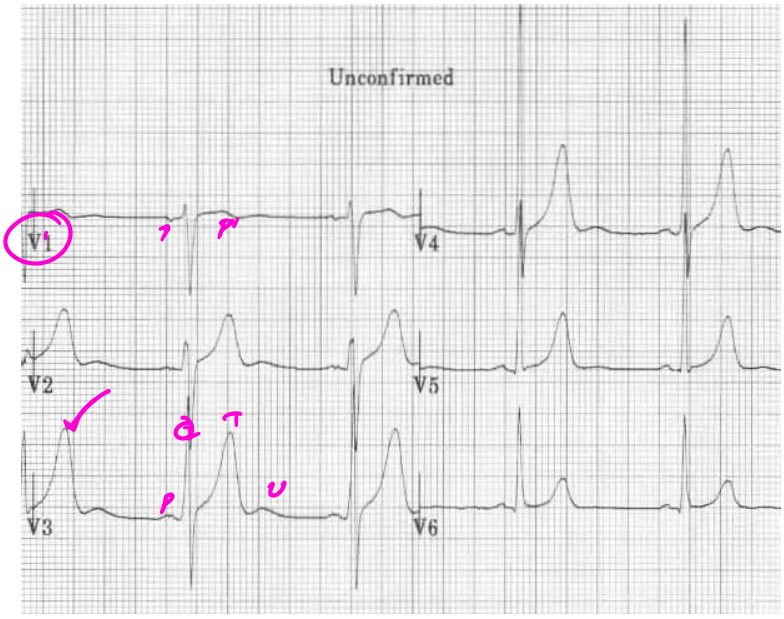
U wave is usually in the same direction as the T wave.
U wave is best seen in leads V2 and V3.
U -wave size is inversely proportional to heart rate: the U wave grows bigger as the heart rate slows down
U waves generally become visible when the heart rate falls below 65 bpm

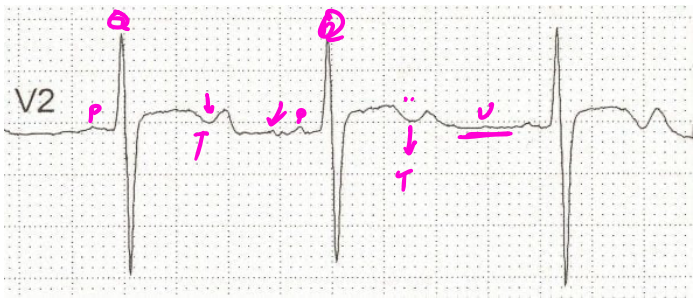


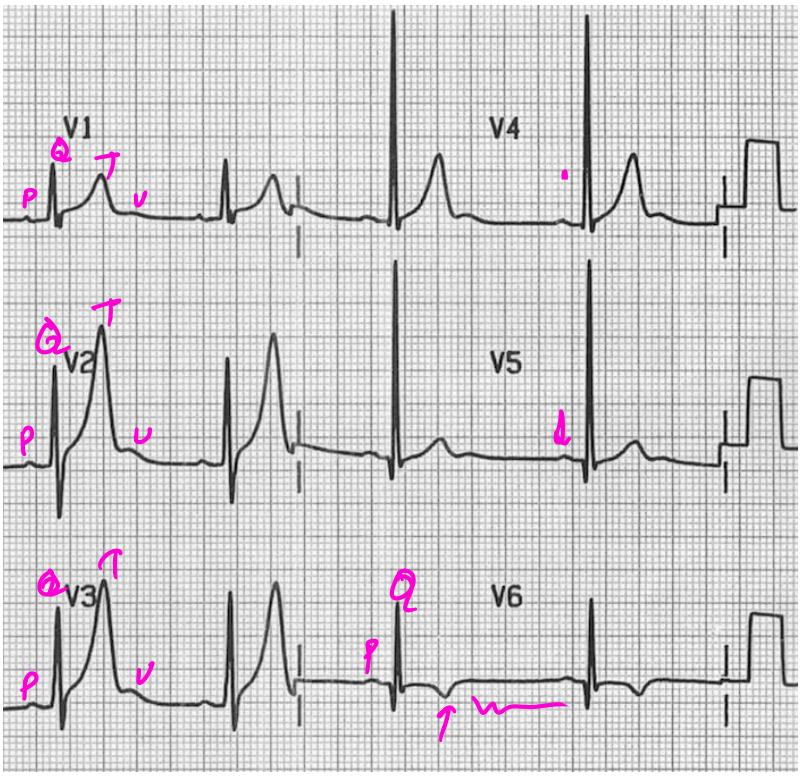

Prominant U waves

Bradycardia
Severe hypokalaemia.




**Prominent U waves due to sinus bradycardia**

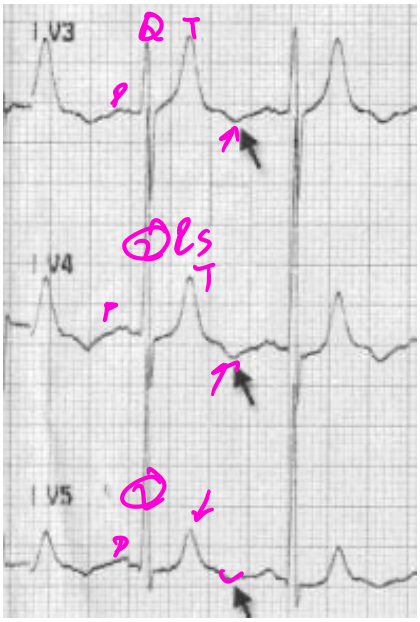


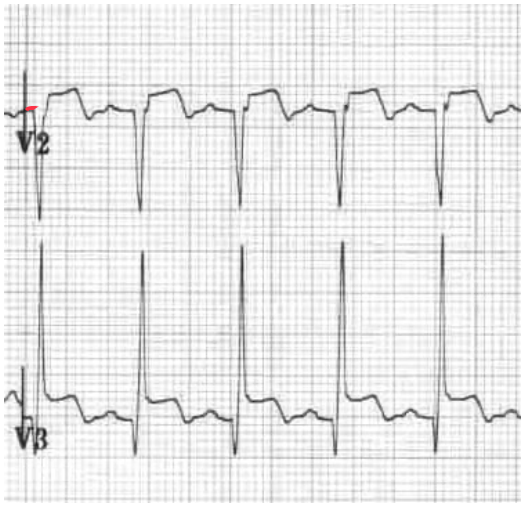
In patients presenting with chest pain, inverted U waves:

Are a very specific sign of myocardial ischaemia

May be the earliest marker of unstable angina and evolving myocardial infarction

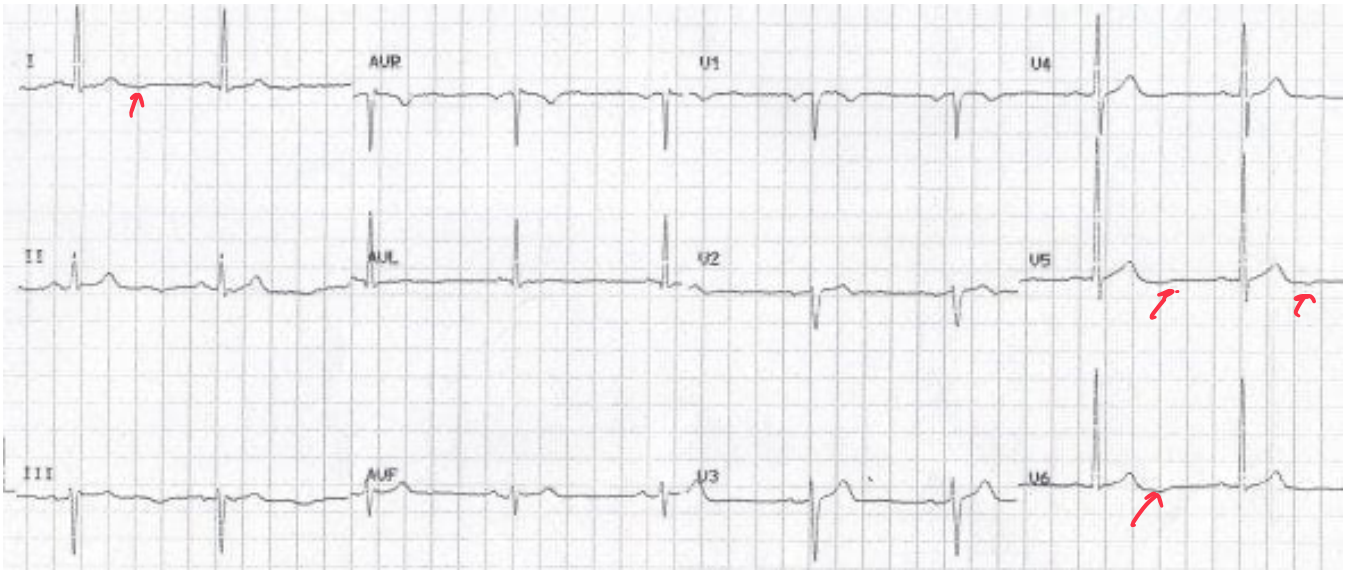
Have been shown to predict a ≥ 75% stenosis of the LAD / LMCA and the presence of left ventricular dysfunction



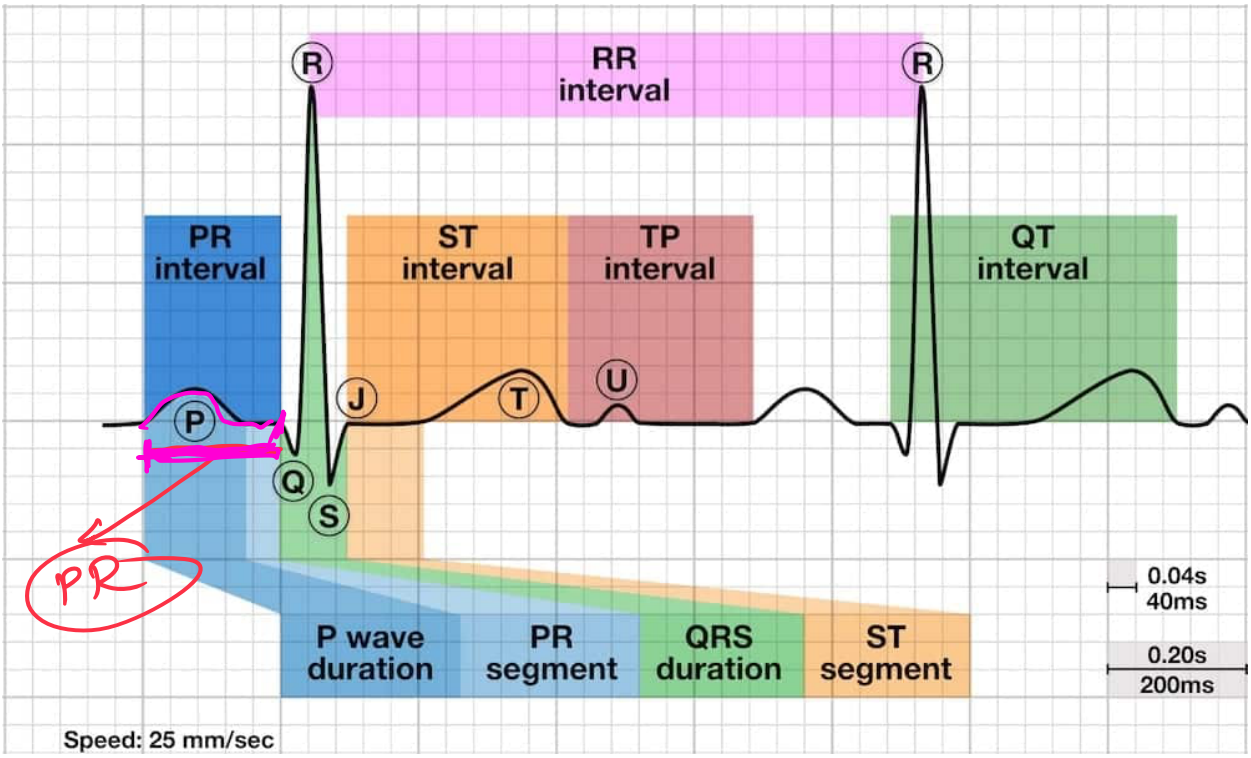

Inverted U waves in Prinzmetal angina

Inverted U waves in a patient with unstable angina. Reproduced from Girish et al.




Note the subtle U-wave inversion in the lateral leads (I, V5 and V6) in this patient with a NSTEMI; these were the only abnormal findings on his ECG.

# PR INTERVAL




The PR interval is the time from the onset of the P wave to the start of the QRS complex. It reflects conduction through the AV node.

The normal PR interval is between 120 – 200 ms (0.12-0.20s) in duration (three to five small squares).

3small

5small boxes

If the PR interval is > 200 ms, first degree heart block is said to be present. PR interval < 120 ms suggests pre-excitation (the presence of an accessory pathway between the atria and ventricles) or AV nodal (junctional) rhythm.

<3 small

1Large box

Prolonged PR Interval – AV block (PR >200ms

>1Large

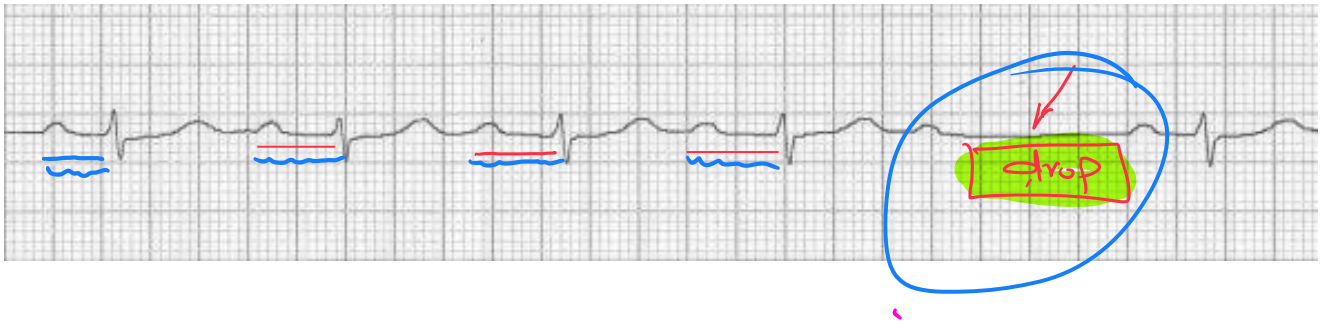
>200

6 boxes

6x40

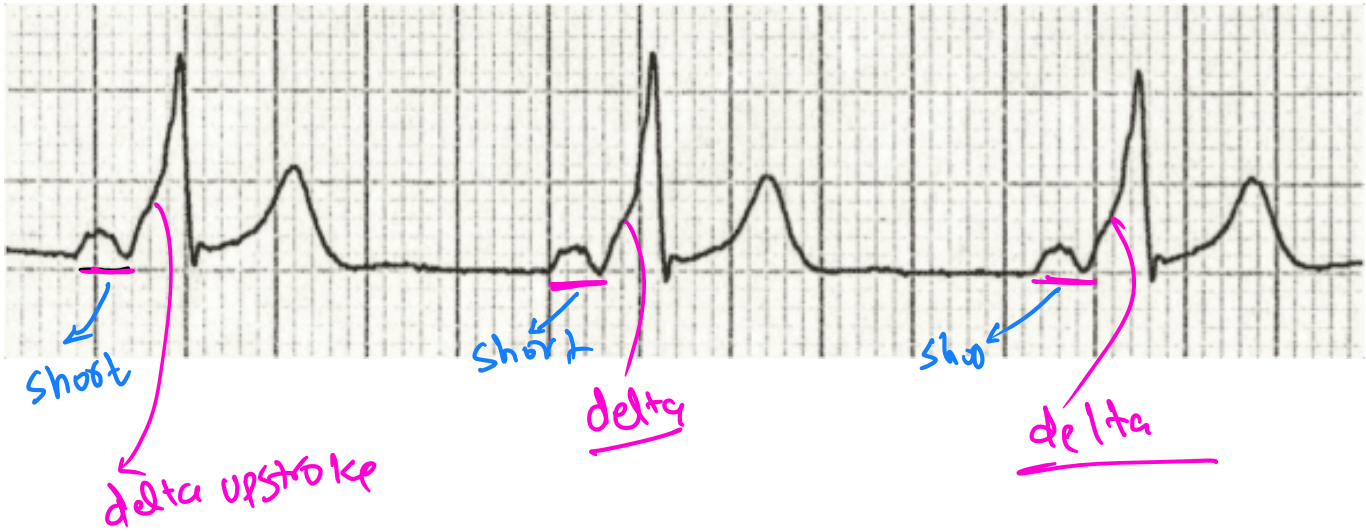
=240

Sinus rhythm with marked 1st degree heart block (PR interval 340ms)




**Second degree heart block, Mobitz type I (Wenckebach phenomenon).**
**Note how the baseline PR interval is prolonged, and then further prolongs with each successive beat, until a QRS complex is dropped.**
**The PR interval before the dropped beat is the longest (340ms), while the PR interval after the dropped beat is the shortest (280ms).**

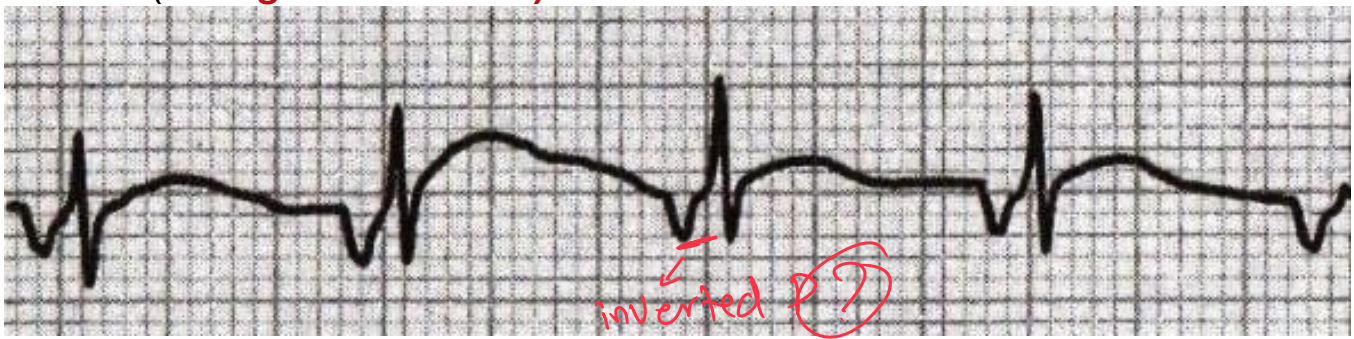
**Short PR interval (<120ms)** → <3 small boxes
**Preexcitation syndromes**
**AV nodal (junctional) rhythm.**



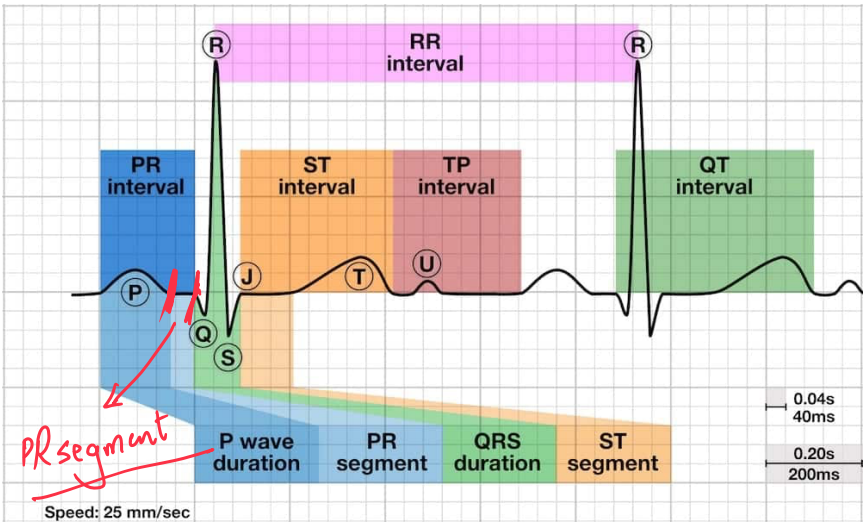

**The characteristic features of Wolff-Parkinson-White syndrome are a short PR interval (<120ms), broad QRS and a slurred upstroke to the QRS complex, the delta wave.**

## AV nodal (junctional) rhythm

Junctional rhythms are narrow complex, regular rhythms arising from the AV node. P waves are either absent or abnormal (e.g. inverted) with a short PR interval (=retrograde P waves).
ECG: Accelerated junctional rhythm demonstrating inverted P waves with a short PR interval (**retrograde P waves**)



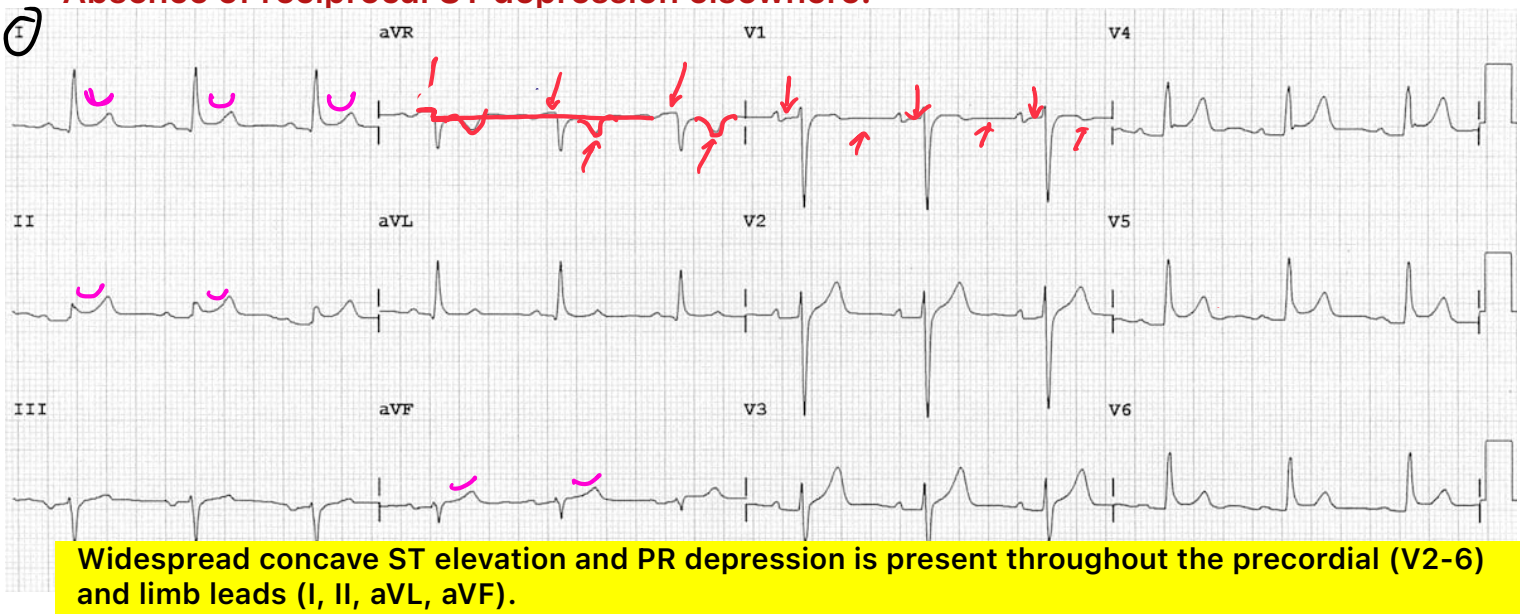
# PR segment abnormalities



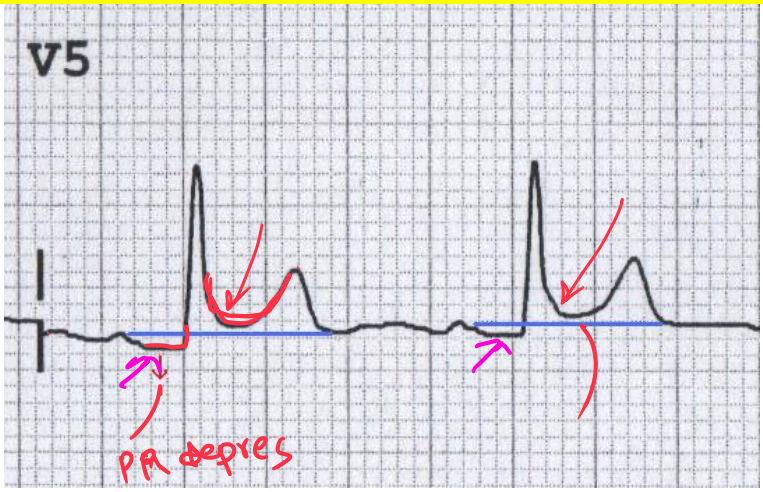

## Pericarditis

The characteristic changes of acute pericarditis are:

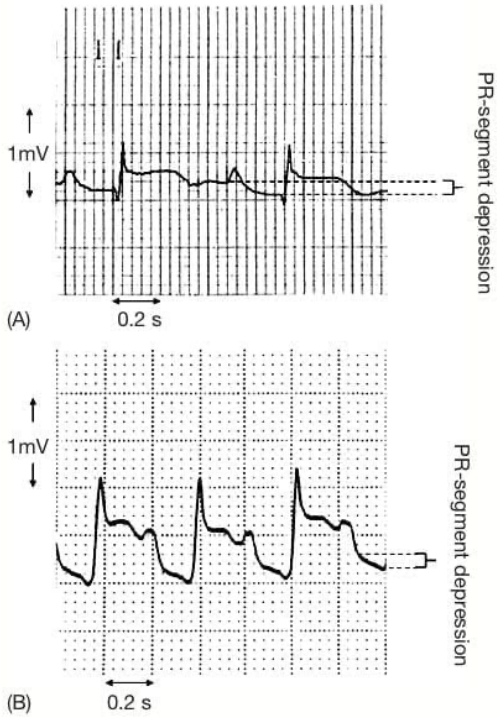
PR segment depression.
Widespread concave ('saddle-shaped') ST elevation.
Reciprocal ST depression and PR elevation in aVR and V1
Absence of reciprocal ST depression elsewhere.

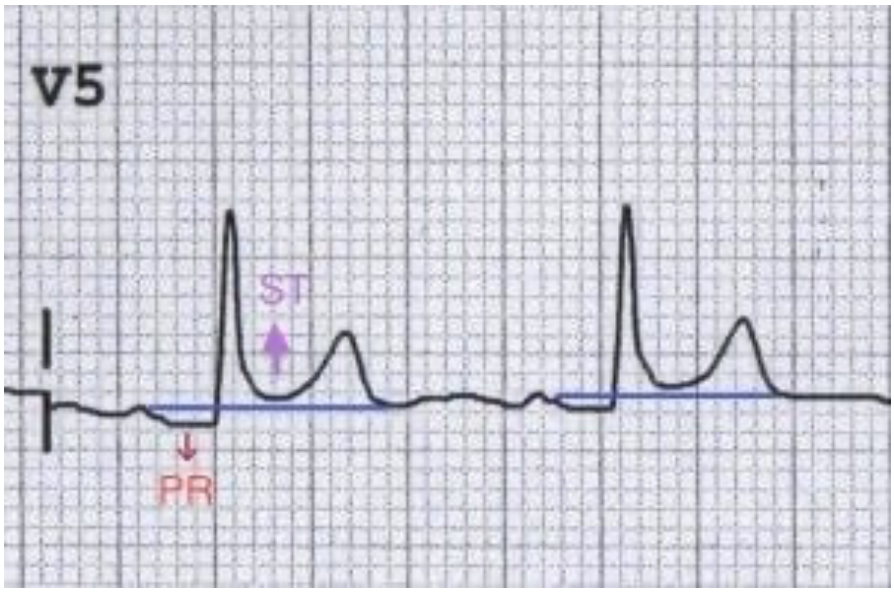



Widespread concave ST elevation and PR depression is present throughout the precordial (V2-6) and limb leads (I, II, aVL, aVF).

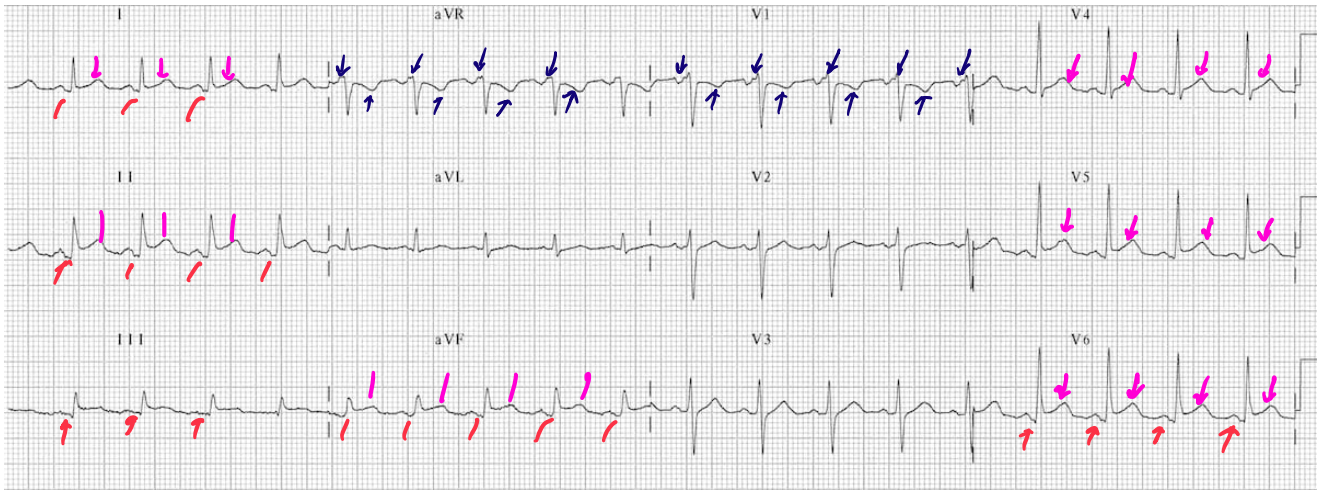



PR segment depression in V5 due to acute pericarditis (note also some concave ST elevation)

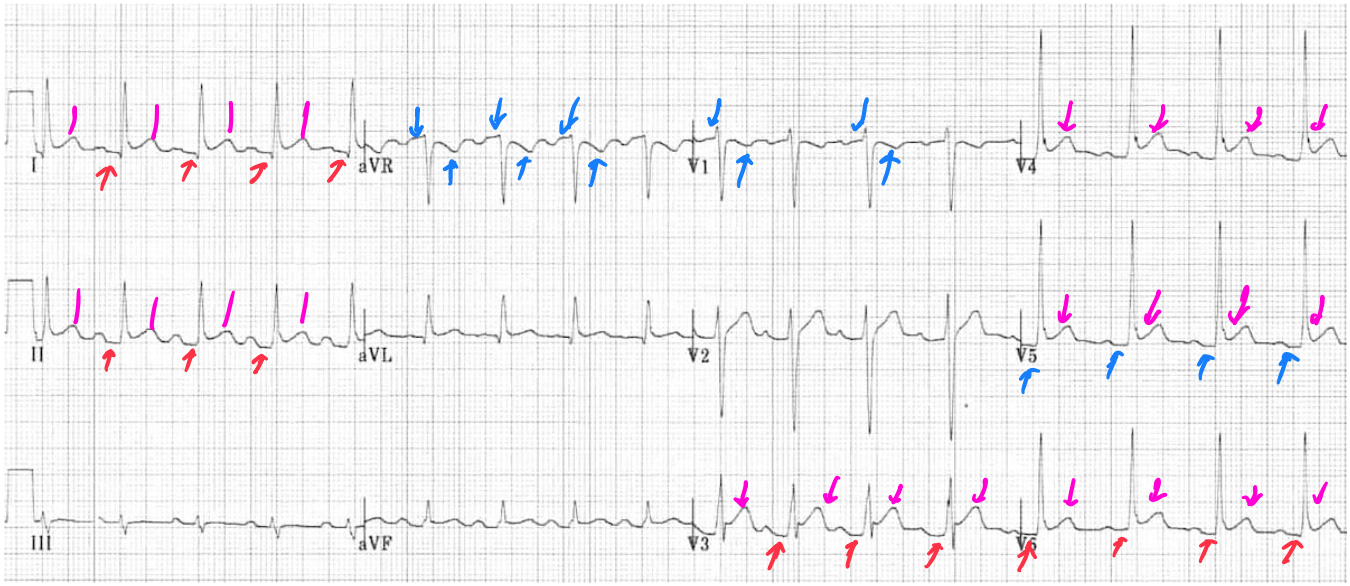


**Reciprocal PR elevation and ST depression in aVR**




Sinus tachycardia
Widespread concave STE and PR depression (I, II, III, aVF, V4-6)
Reciprocal ST depression and PR elevation in V1 and aVR
Spodick's sign best visualised in lead II

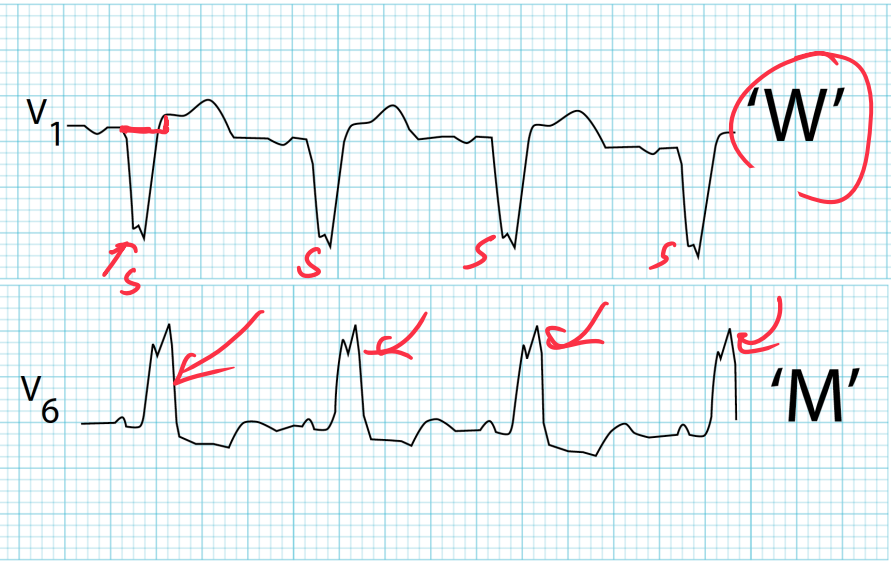



Widespread ST elevation and PR depression
Reciprocal ST depression and PR elevation in V1 and aVR

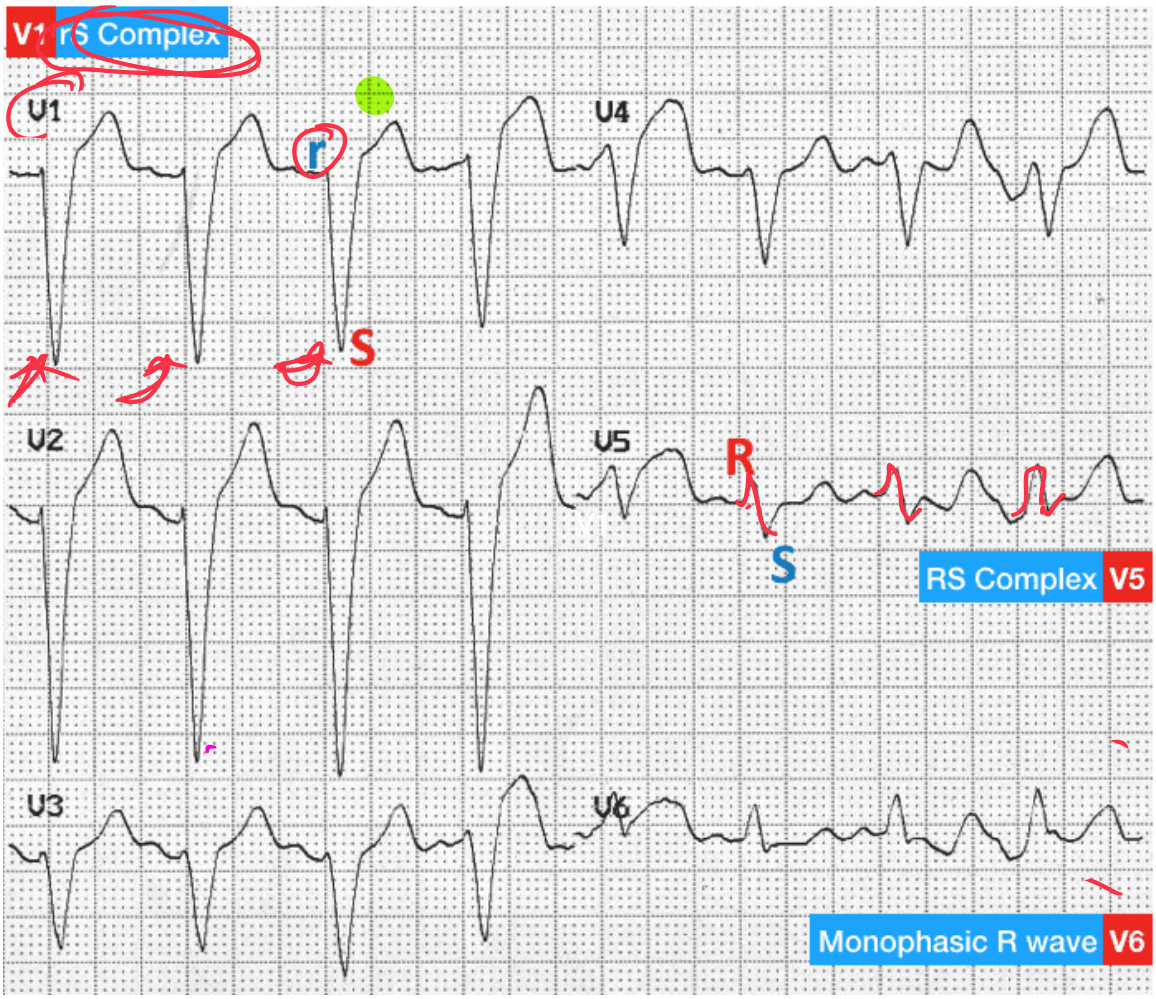
## Left Bundle Branch Block (LBBB)

>3small box

QRS duration ≥ 120ms
Dominant S wave in V1
Broad monophasic R wave in lateral leads (I, aVL, V5-6)
Absence of Q waves in lateral leads
Prolonged R wave peak time > 60ms in leads V5-6

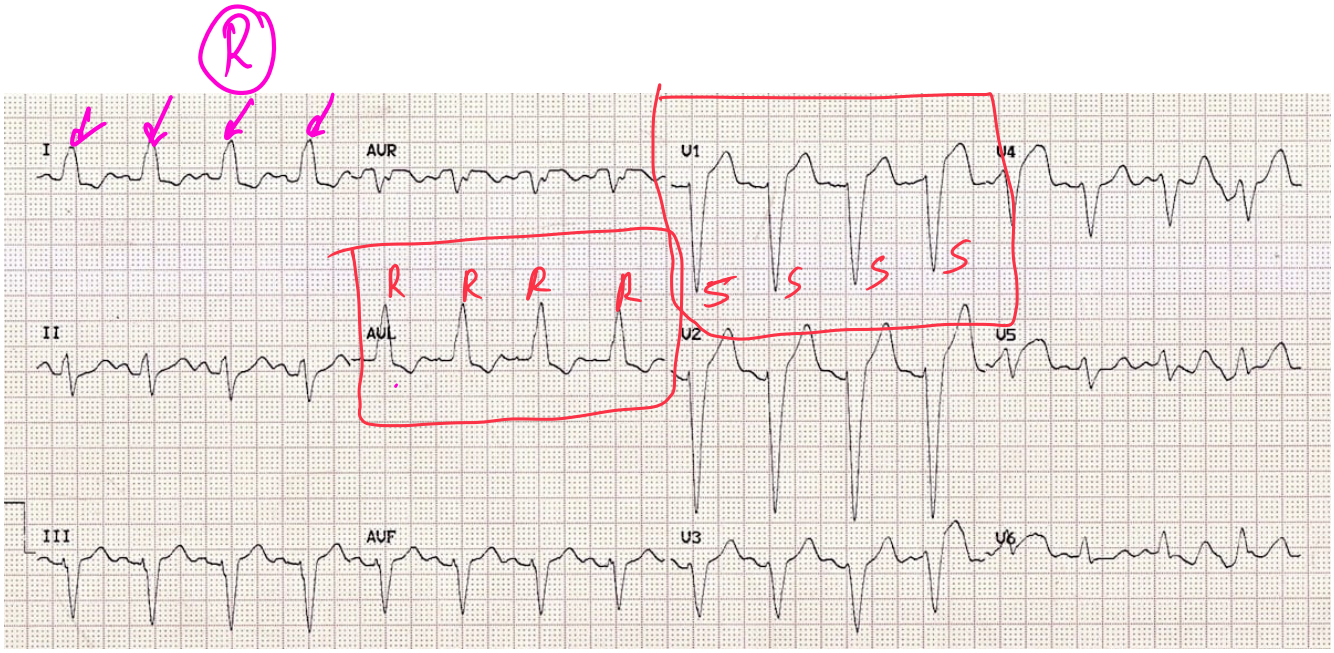



V1: Dominant S wave
V6: broad, notched ('M'-shaped) R wave

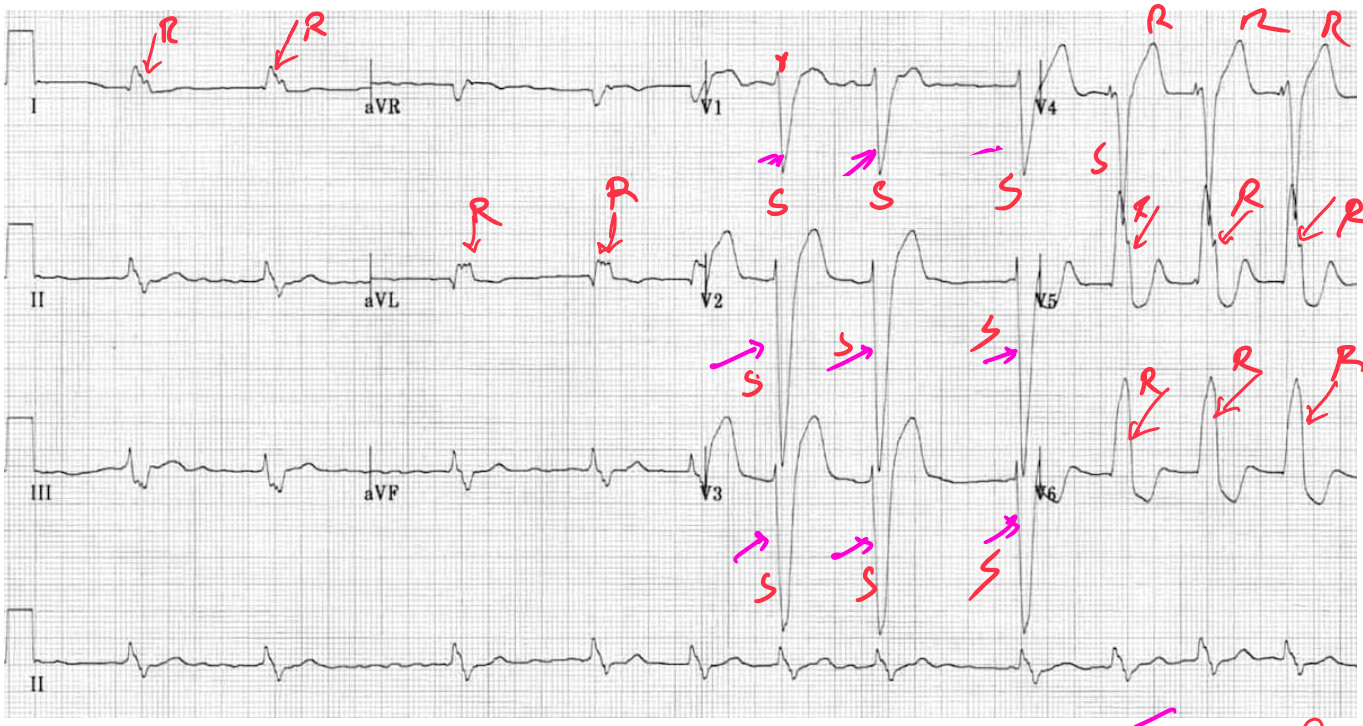



**Left bundle branch block (LBBB):**

1) rS complex in V1 (tiny R wave, deep S wave)
2) Characteristic lateral lead morphology in V5-6
3) Note appropriate discordance in V1 with ST elevation and upright T wave

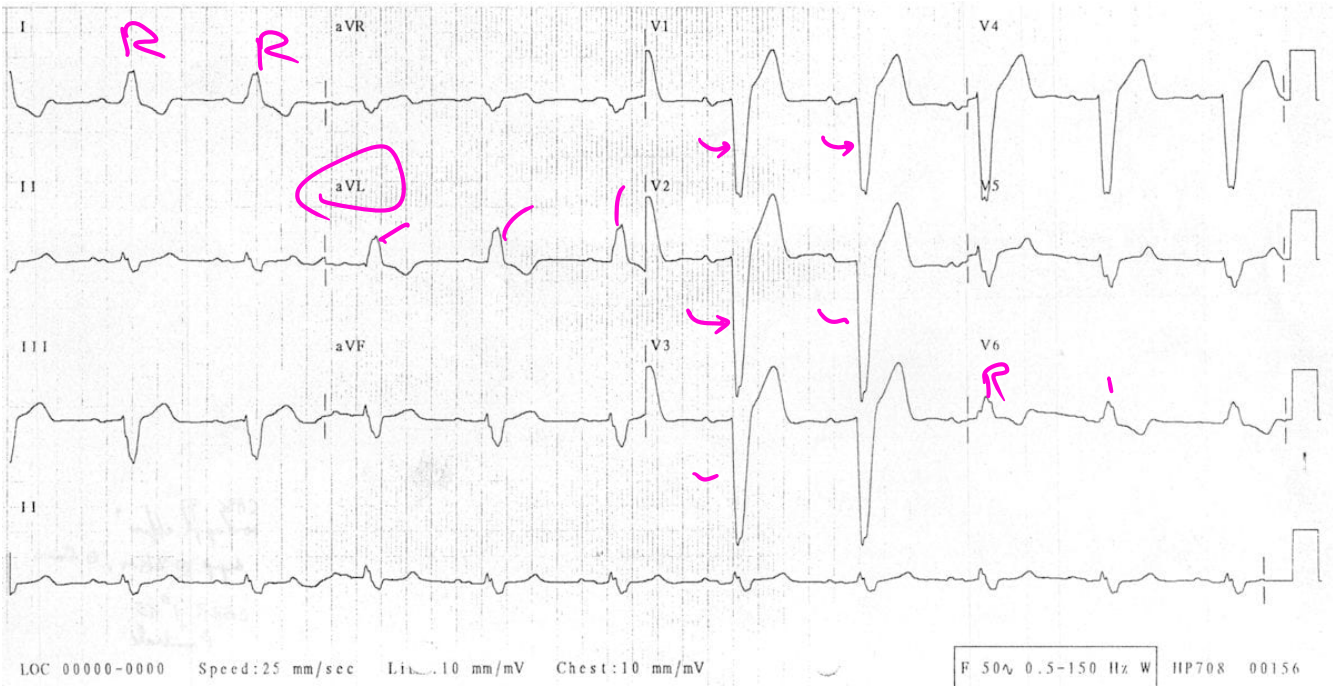


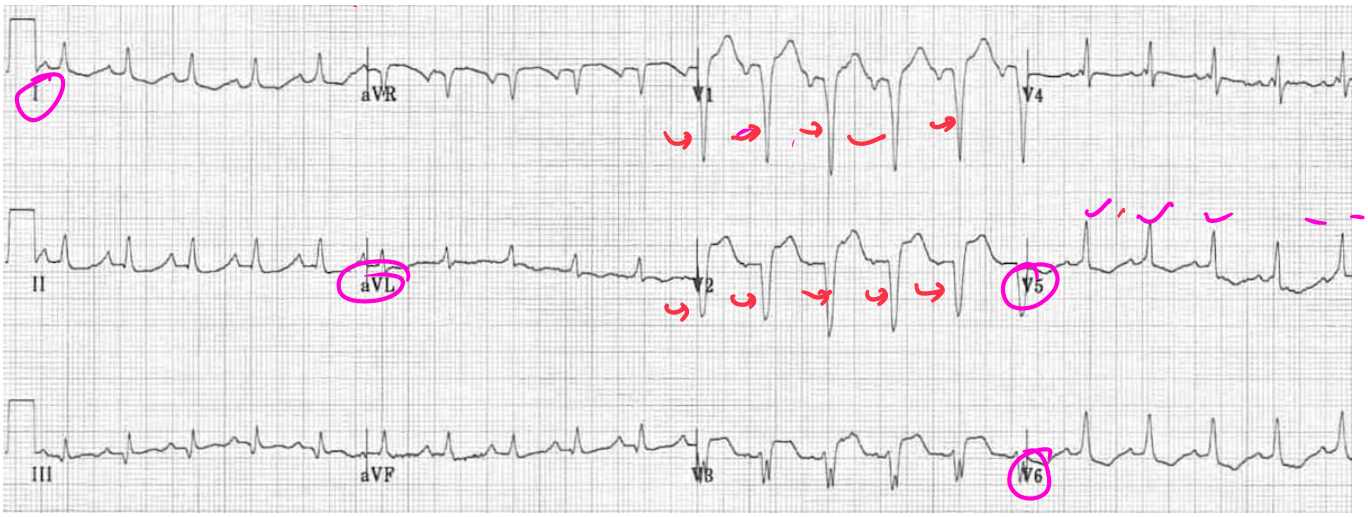

Broad notched R waves are best appreciated in leads aVL and I here. There is absence of Q waves in leads V5-6.




Red → R wave
Pink → S waves

**LBBB with AF.** Note deep S waves in leads V1-3 and tall broad R waves laterally. Appropriate discordance is present.






**Incomplete LBBB is diagnosed when typical LBBB morphology is associated with a QRS duration <120ms.**

## Right Bundle Branch Block (RBBB)

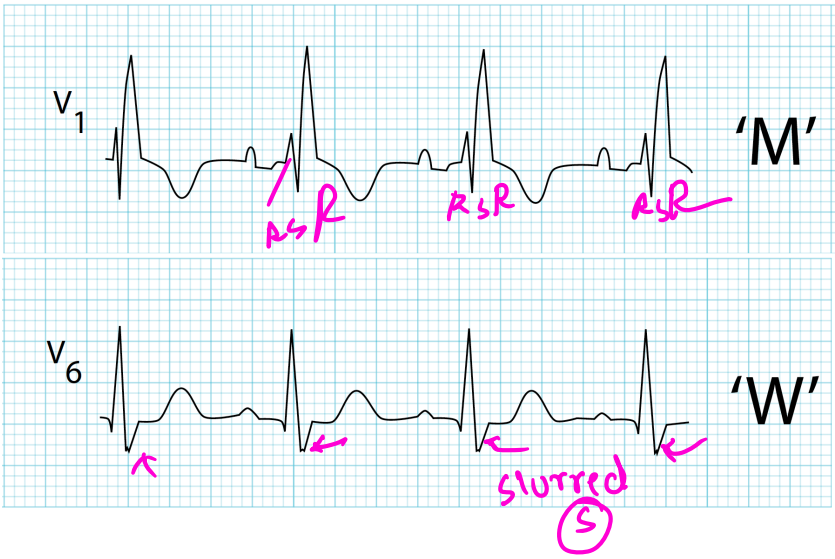
QRS duration > 120ms
RSR′ pattern in V1-3 ("M-shaped" QRS complex)
Wide, slurred S wave in lateral leads (I, aVL, V5-6)

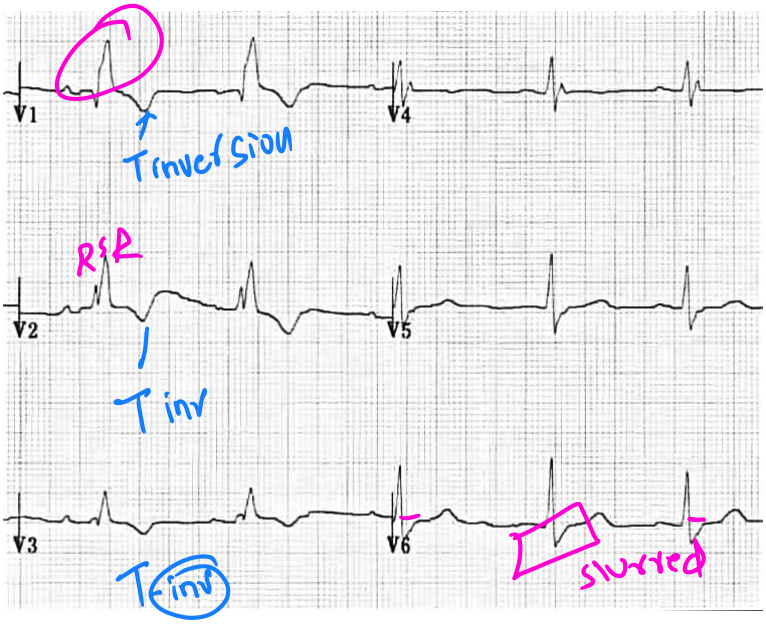
QRS duration ≥ 120ms
Dominant S wave in V1
Broad monophasic R wave in lateral leads (I, aVL, V5-6)
Absence of Q waves in lateral leads
Prolonged R wave peak time > 60ms in leads V5-6

LBBB

MarroW

RSR + Wide S



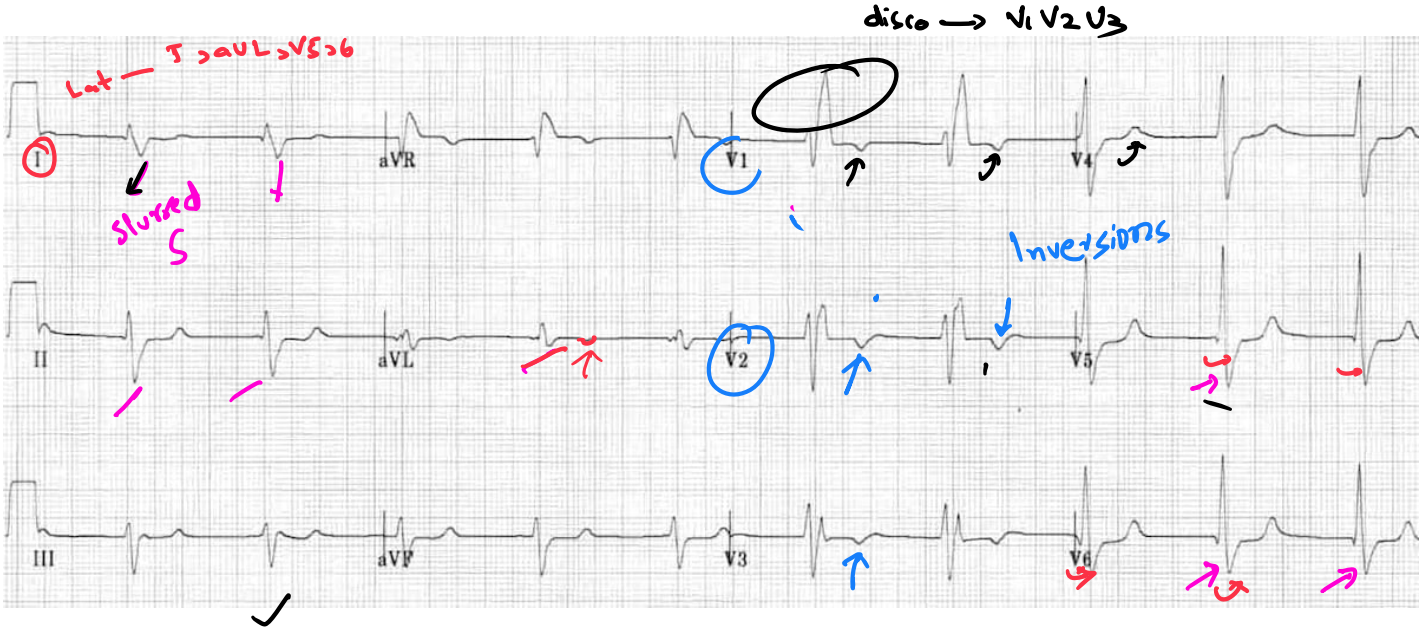



Typical pattern of T-wave inversion in V1-3 with RBBB

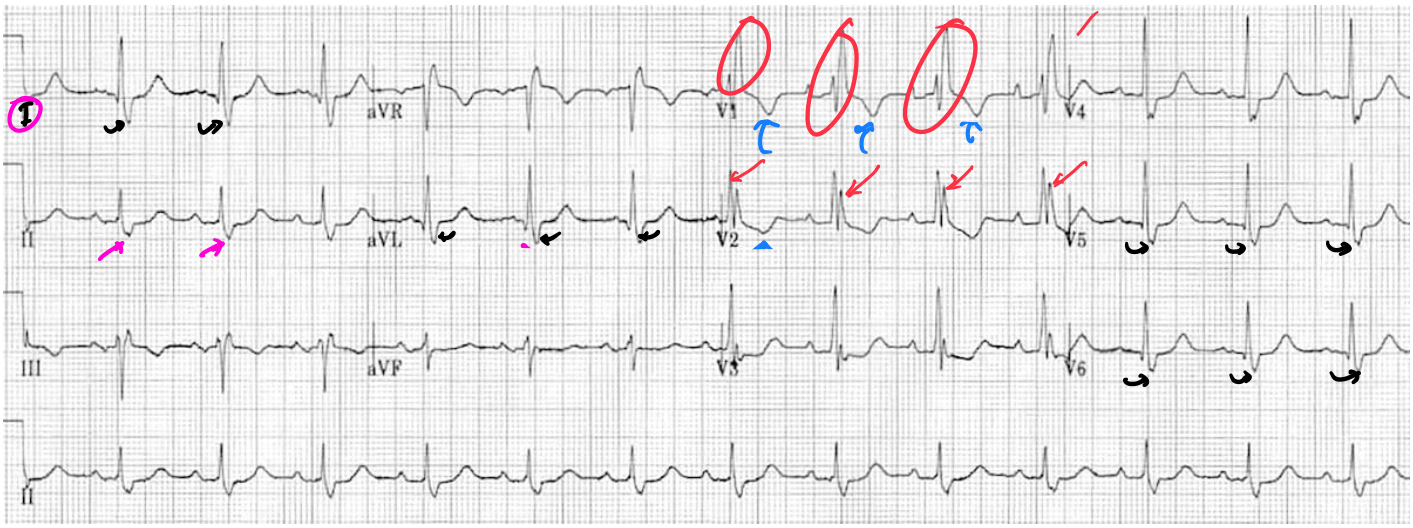
## Appropriate discordance

Appropriate discordance refers to the fact that **abnormal depolarisation should be followed by abnormal repolarisation**, which appears discordant to the preceding QRS complex

In RBBB, this manifests as **ST depression and/or T-wave inversion in leads V1-3**

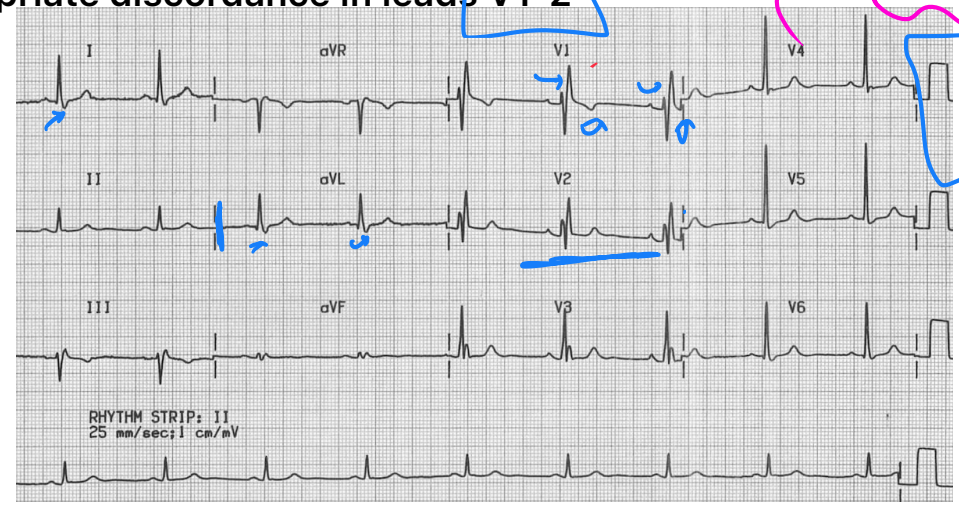



Broad QRS > 120ms
Note the prominent delayed RV conduction, manifested as a tall, broad R wave (R')
best seen in lead V1
Widened S wave is best appreciated in lead I
There is appropriate discordance in the right precordial leads with T-wave inversion




Isolated RBBB.

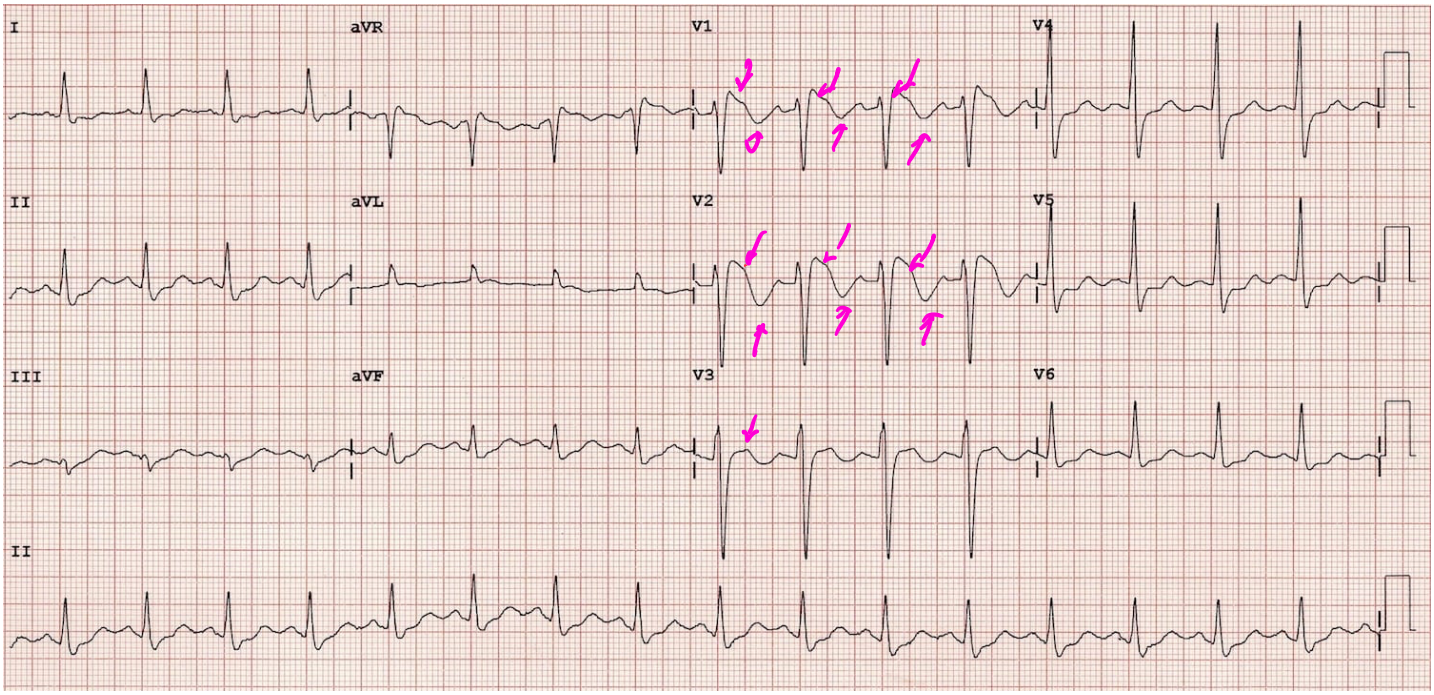
Typical RSR' pattern in V1-2
Widened S waves again demonstrated in lateral leads, especially V4-6
Appropriate discordance in leads V1-2

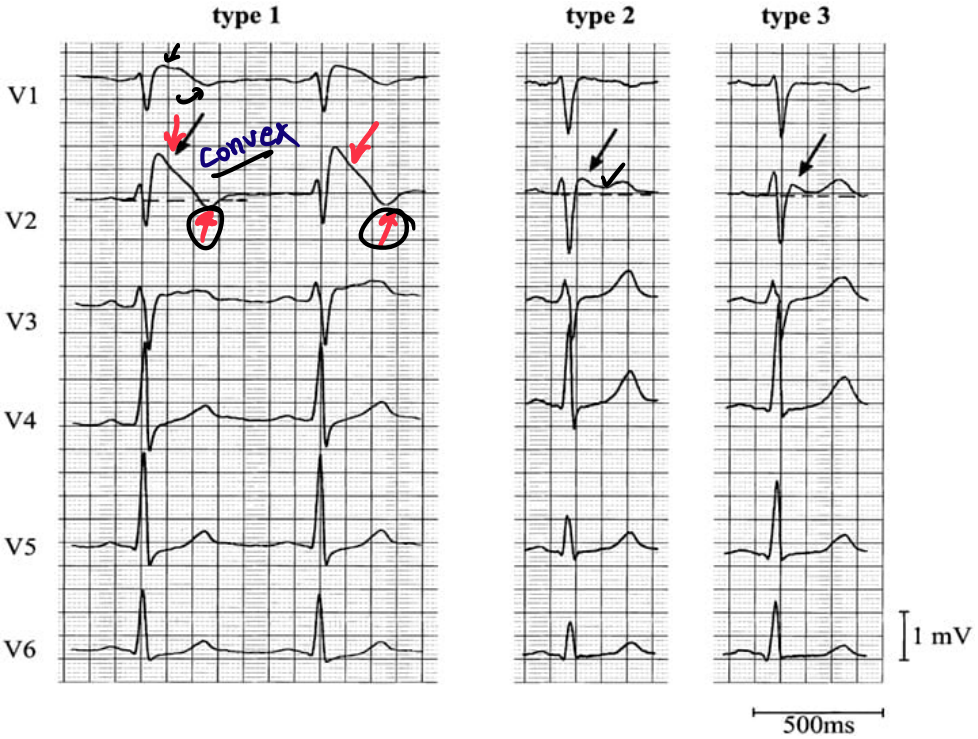


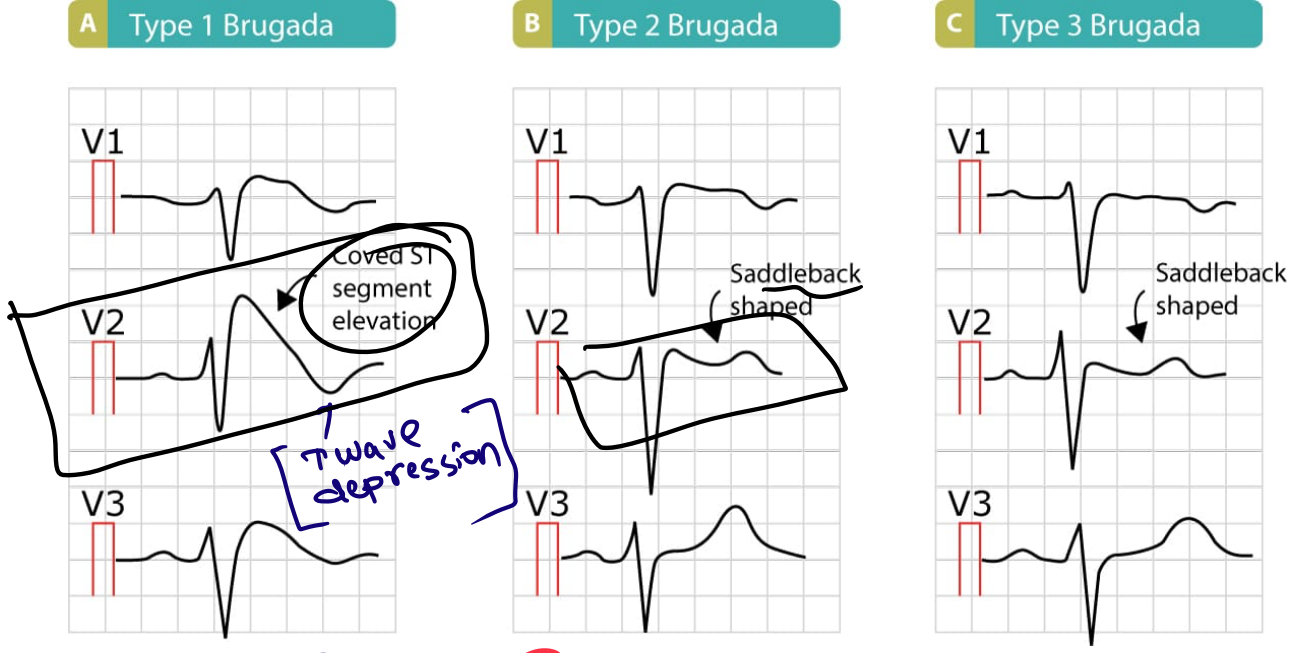
Isolated RBBB.

RSR′ pattern in V1-3
Lateral S wave changed are not evident here
Note note normal axis in isolated RBBB

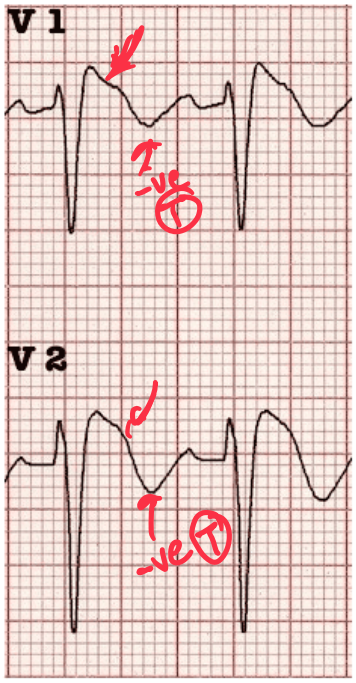
## BRUGADA SYNDROME





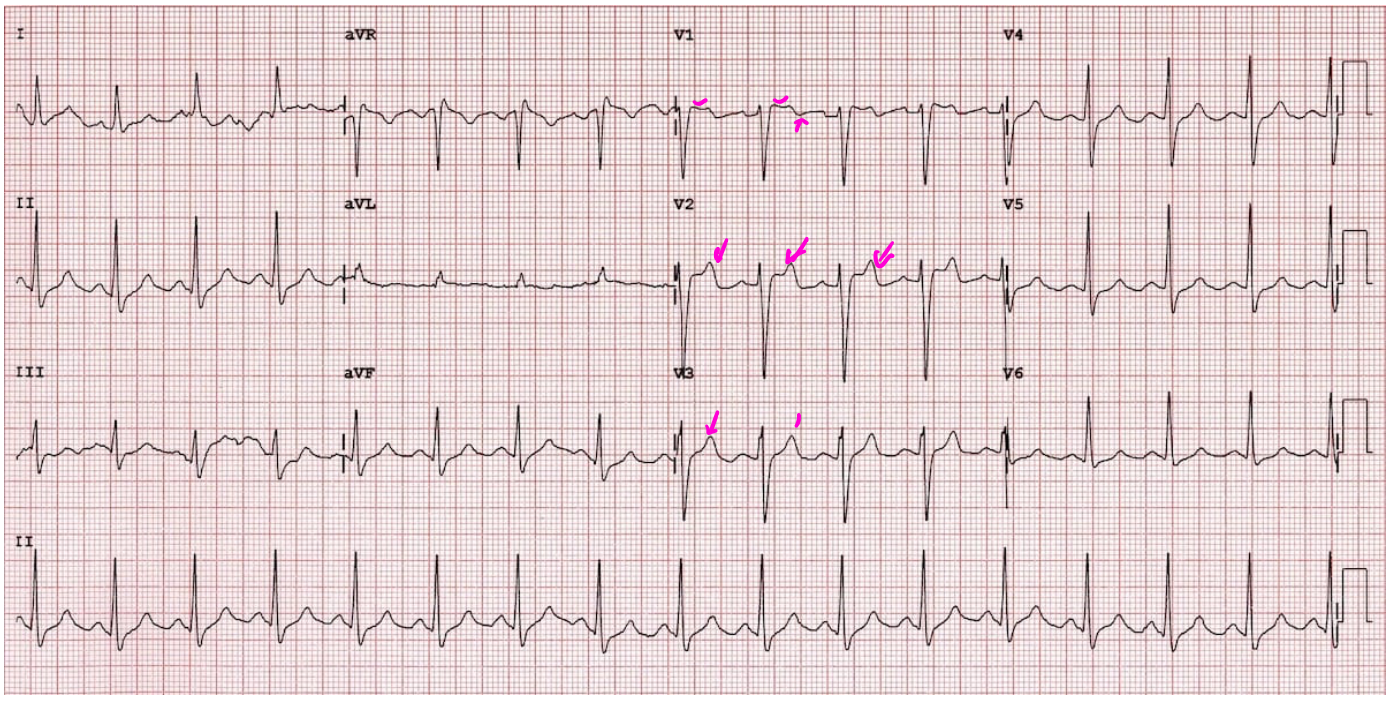

Type (1)



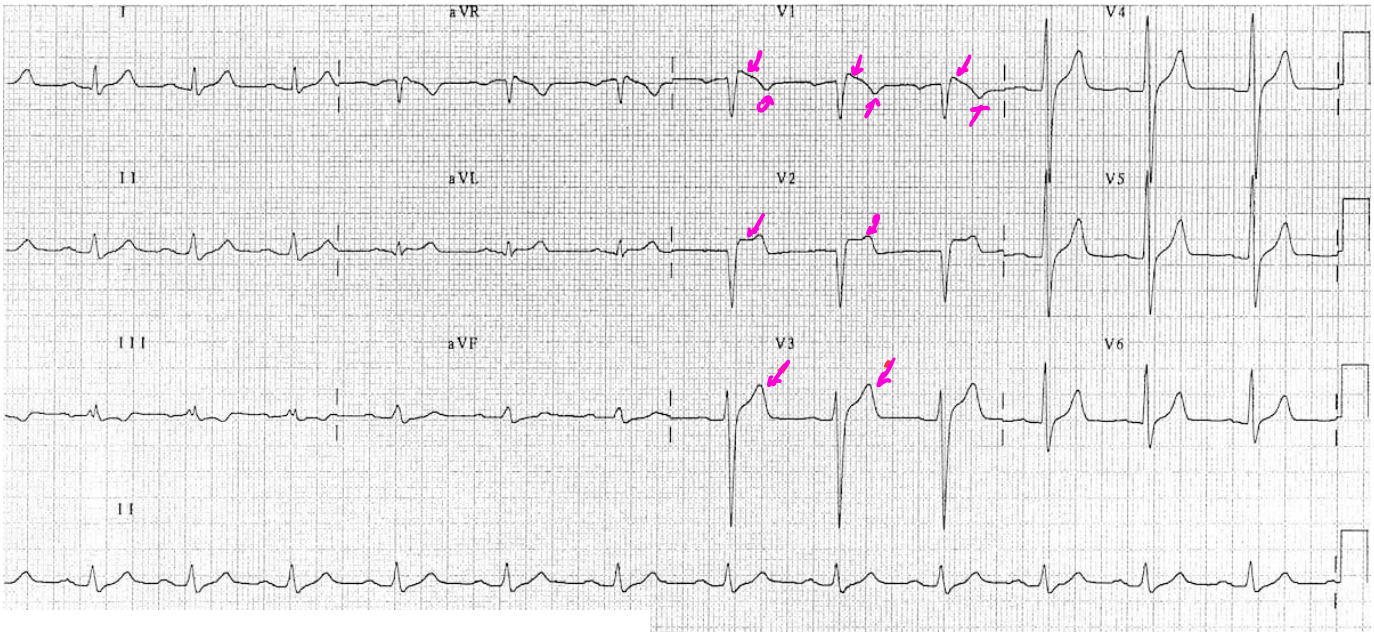

Coved ST segment elevation >2mm in >1 of V1-V3 followed by a negative T wave.
This is the only ECG abnormality that is potentially diagnostic.
It is often referred to as Brugada sign.

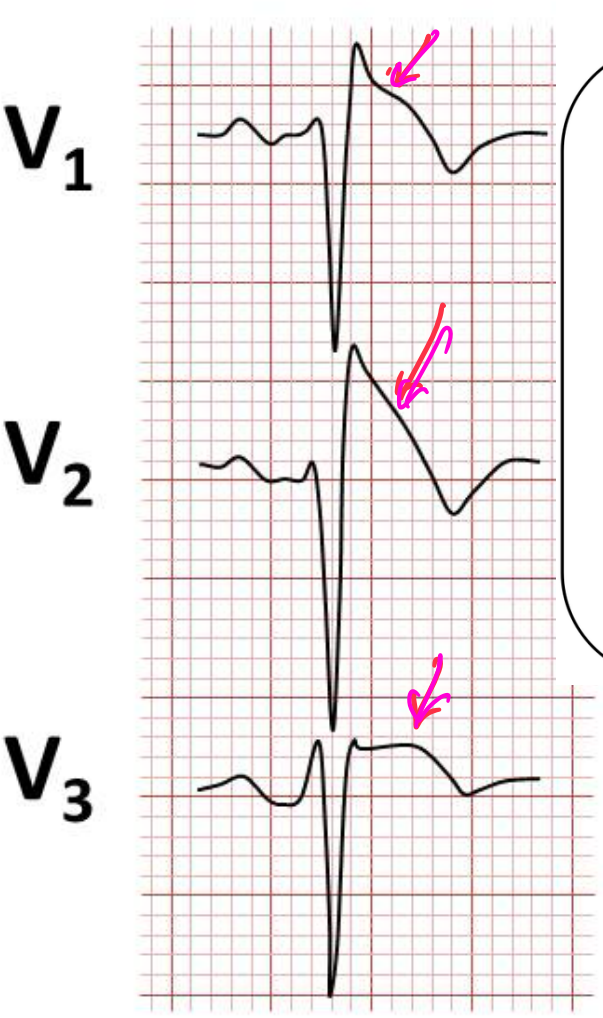
$V_1 V_2 V_3 \rightarrow \geq 1$

Type 2
Brugada Type 2 has >2mm of saddleback shaped ST elevation.



**Brugada type 3: can be the morphology of either type 1 or type 2, but with <2mm of ST segment elevation.**



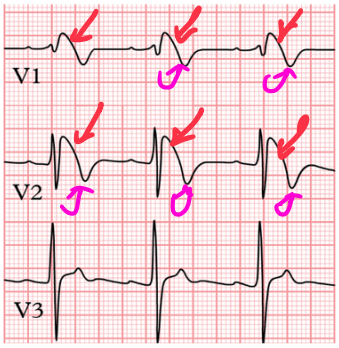




EMNote

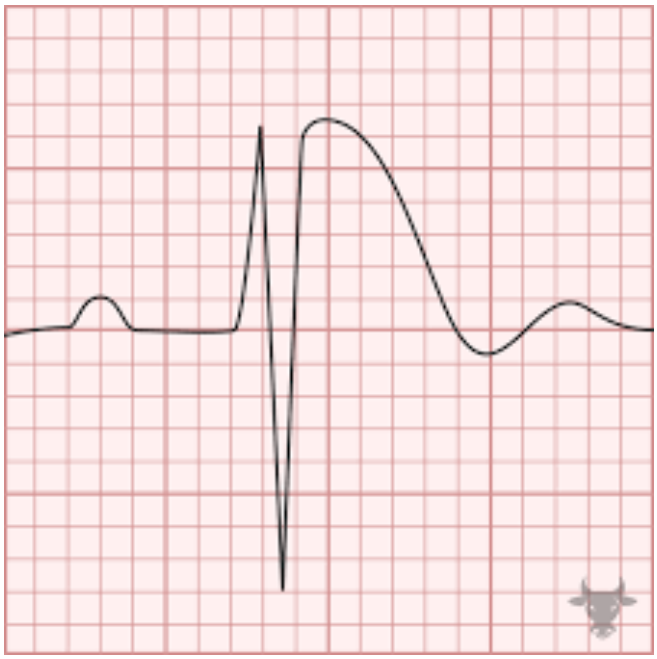
## Brugada Syndrome

1. Synonyms:
   - Pokkuri Death Syndrome
   - SUNDS: Sudden Unexpected Nocturnal Death Syndrome
2. Na channelopathy:
   - Familial clustering, autosomal dominant inheritance
3. Manifestations:
   - Syncope, VT, VF, sudden death at sleep
4. Treatment:
   - ICD (Implantable cardioverter-defibrillator ), quinidine
5. Diagnostic ECG

(coved type STE & TWI in V1~3)

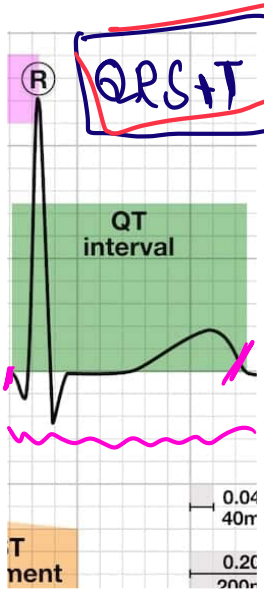



@ jackcfchong

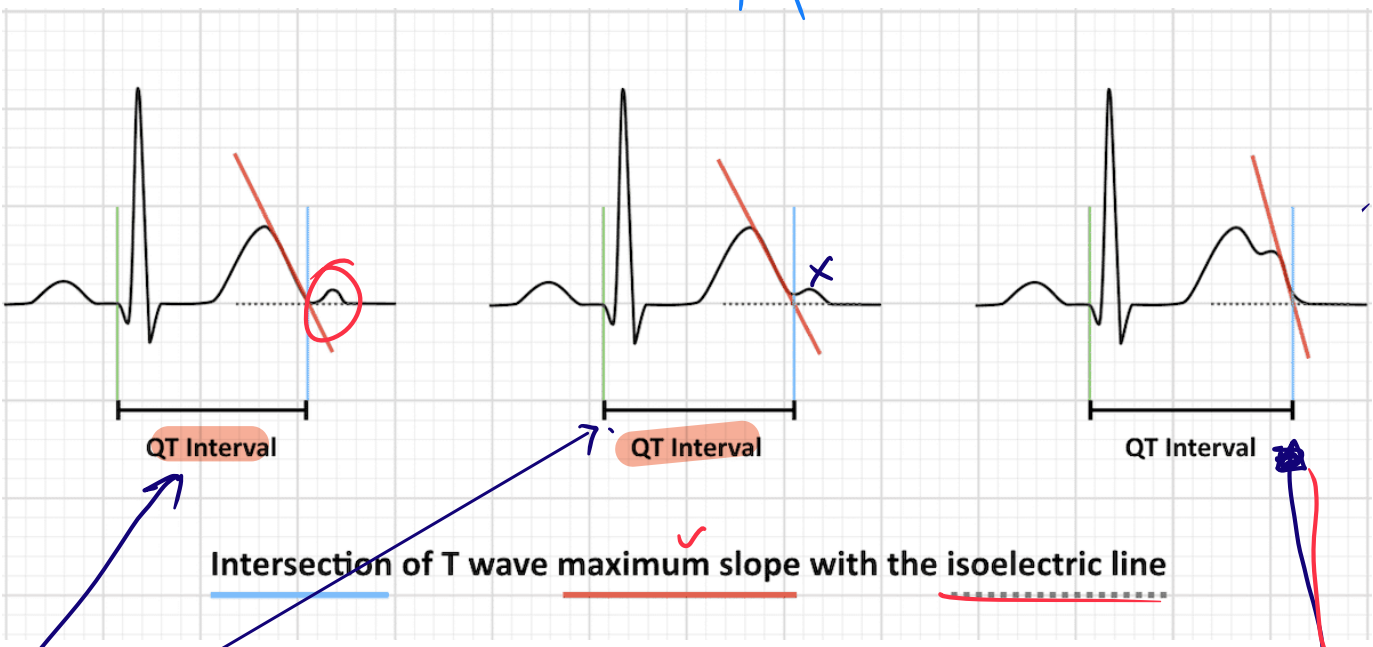


# QT PROLONGATION

Time from the start of the Q wave to the end of the T wave
Represents time taken for ventricular depolarisation and repolarisation, effectively the period of ventricular systole from ventricular isovolumetric contraction to isovolumetric relaxation

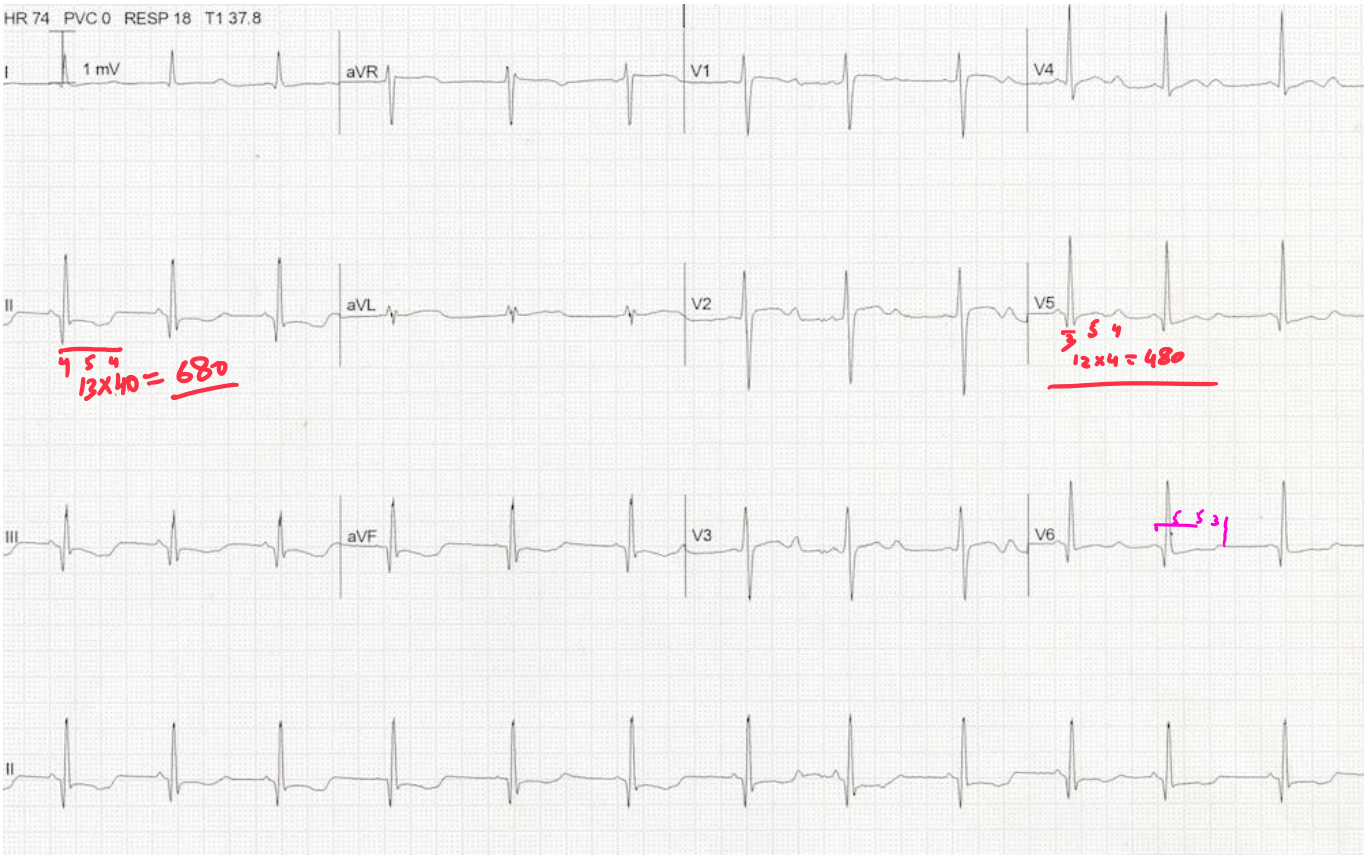



The QT interval is usually measured in either lead II or V5-6, however the lead with the longest measurement should be used
Several successive beats should be measured, with the maximum interval taken
Large U waves (> 1mm) that are fused to the T wave should be included in the measurement
Smaller U waves and those that are separate from the T wave should be excluded
The maximum slope intercept method is used to define the end of the T wave (see below)

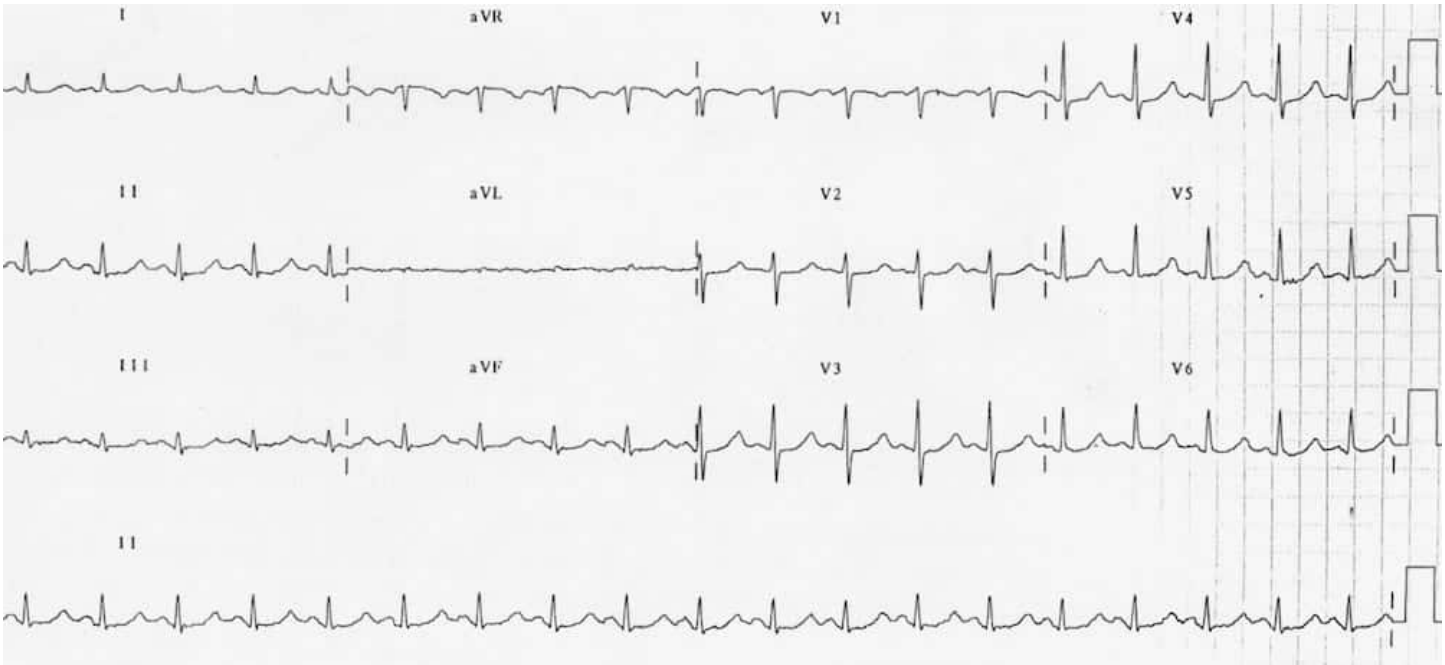


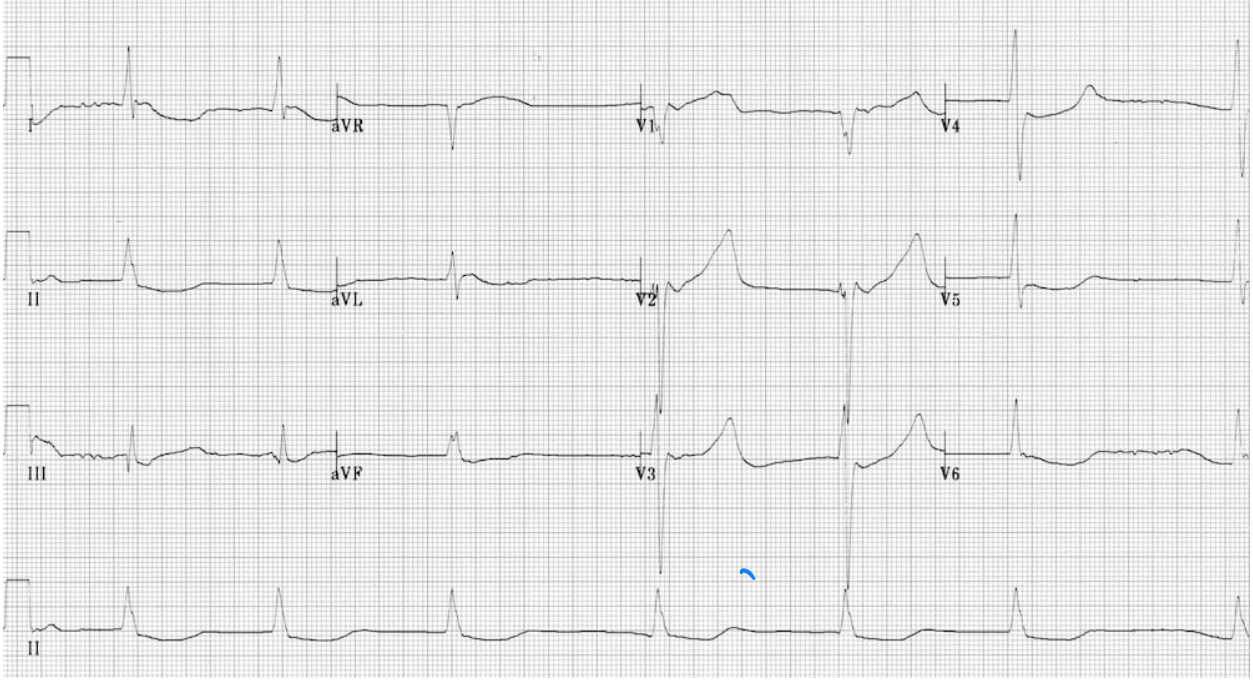

Left, middle: Smaller U waves and those that are separate from the T wave should be excluded from measurements

Right: Large U waves that are fused to the T wave should be included in measurements



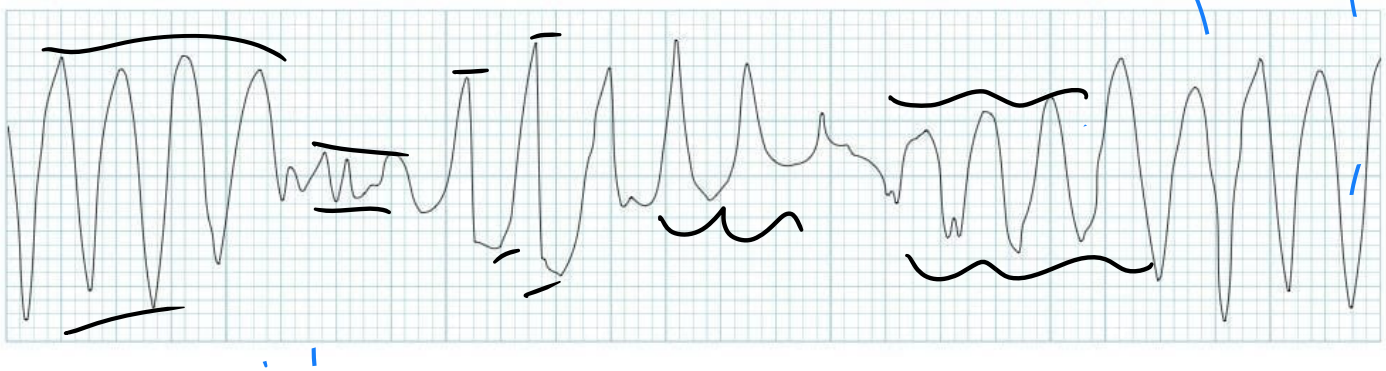

Apparent QTc 500ms
There are **prominent U waves in precordial leads**
This patient had a **K of 1.9**
Hypokalaemia causes apparent QTc prolongation in the limb leads (due to T-U fusion) with prominent U waves in the precordial leads.

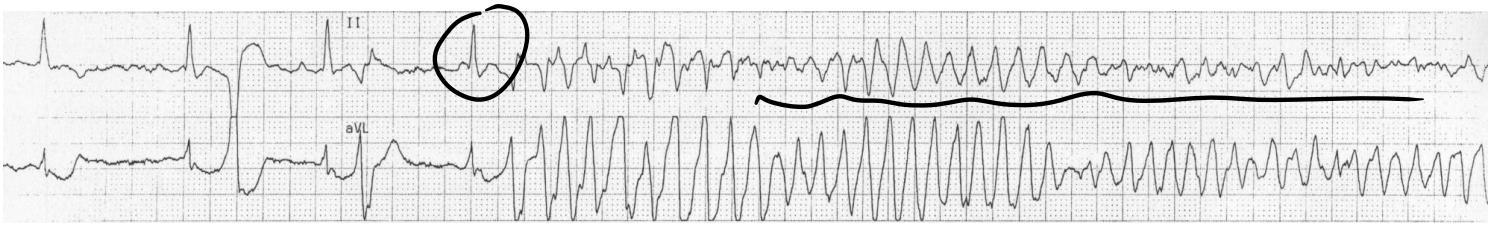


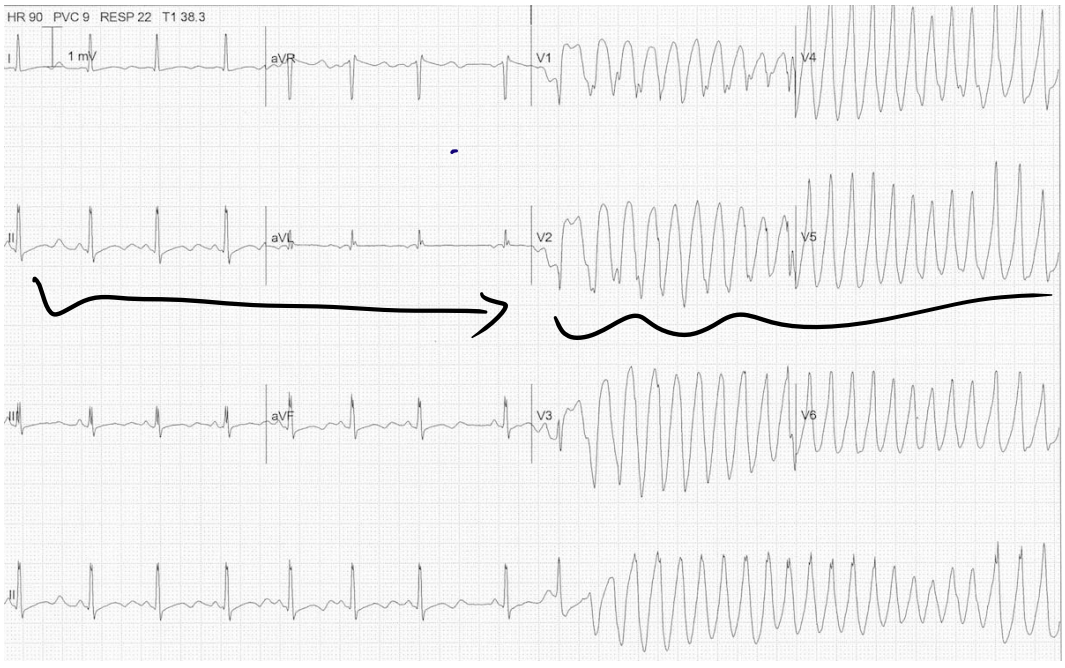



## Torsades de pointes (TdP)

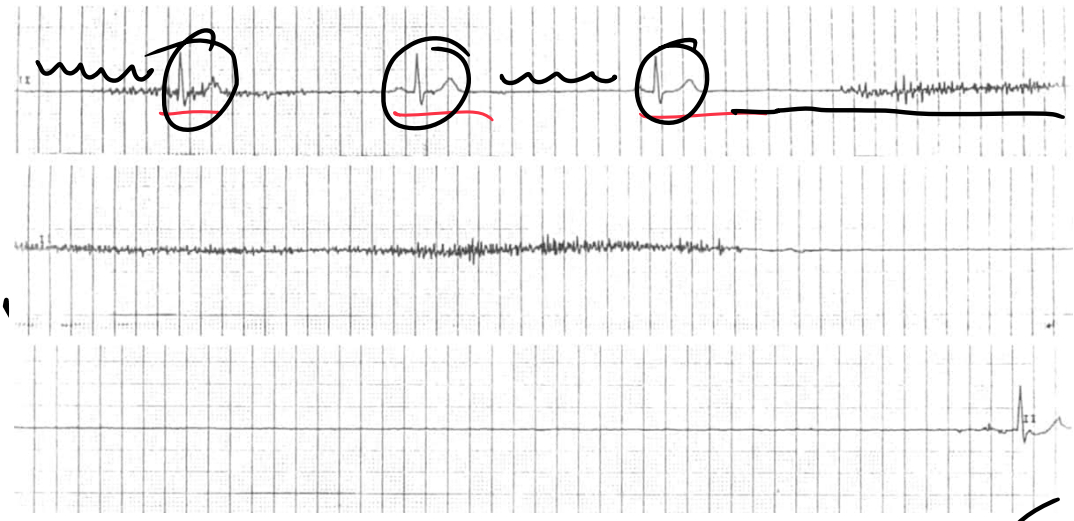
specific form of **PVT** **occurring in the context of QT prolongation** — it has a characteristic morphology **in which the** **QRS complexes "twist" around the isoelectric line.**



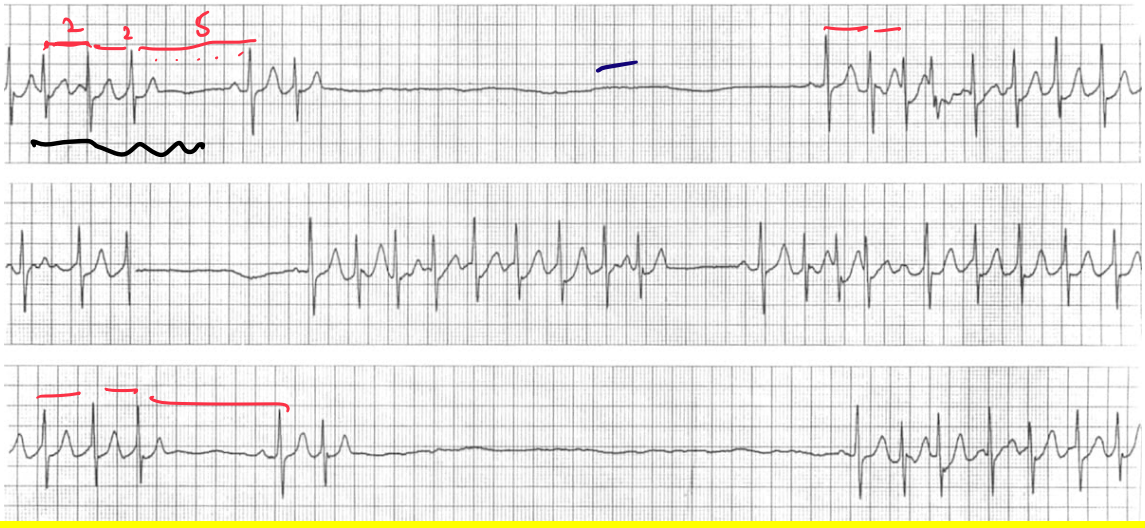


## ECG in Sinus Node Dysfunction



**Sinus arrest:** Prolonged absence of sinus node activity (absent P waves) > 3 seconds.



**Bradycardia-tachycardia syndrome:**
Runs of tachycardia interspersed with long sinus pauses (up to 6 seconds).
The sinus rate is extremely slow, varying from 40 bpm down to around 10 bpm in places.
Sinus beats are followed by paroxysms of junctional tachycardia at around 140 bpm.

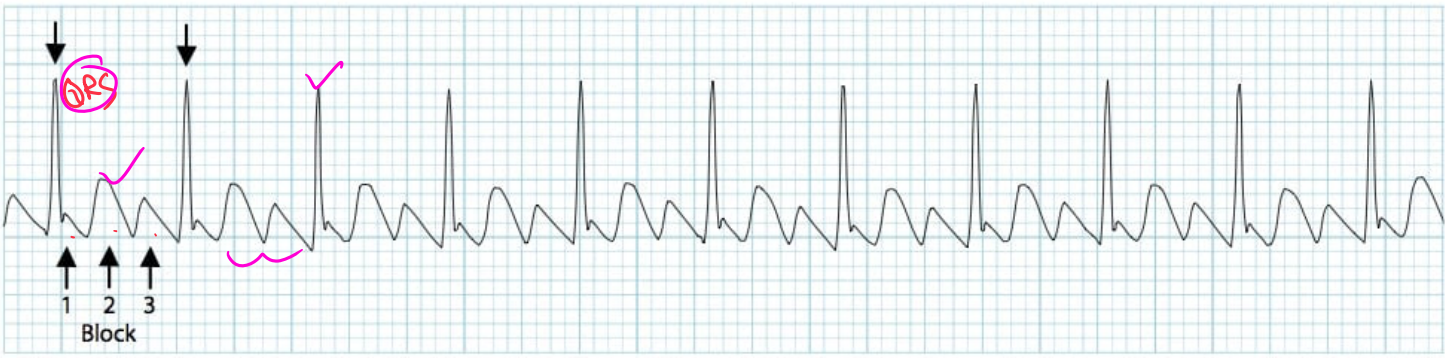
# ATRIAL FLUTTER

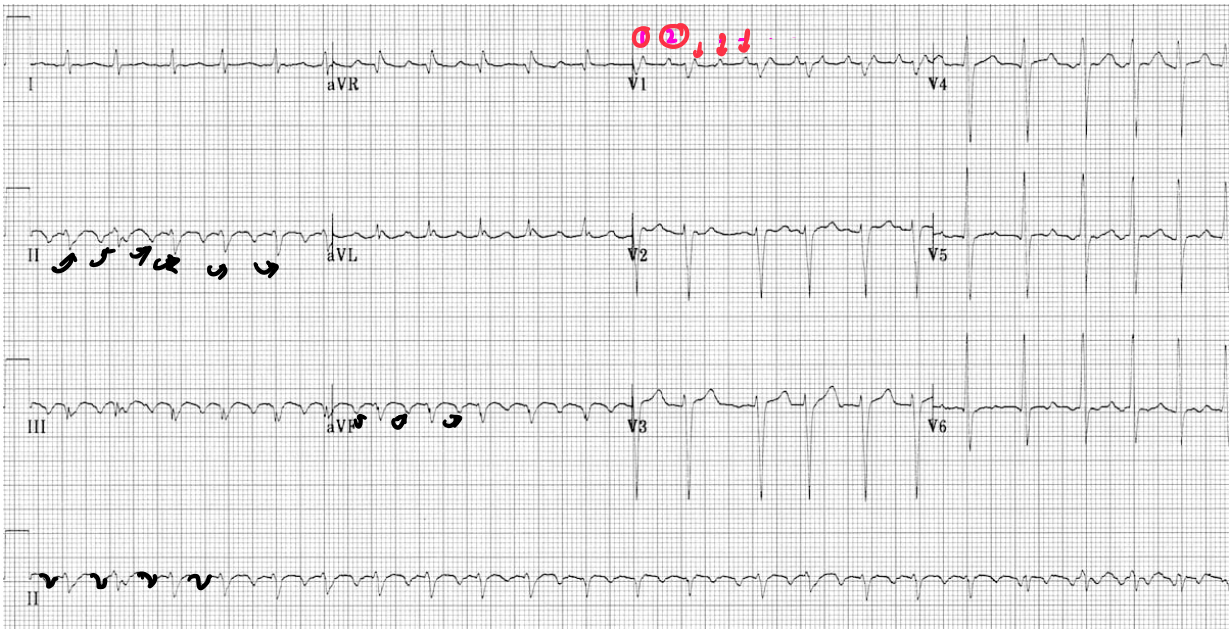
QRS duration: < 120 ms

**Narrow complex tachycardia**
Regular **atrial activity at ~300 bpm**
Loss of the isoelectric baseline
"**Saw-tooth**" pattern of inverted flutter waves in **leads II, III, aVF**
**Upright flutter waves in V1 that may resemble P waves**
Ventricular rate depends on AV conduction ratio (see below)

**Fixed AV conduction ratio ("AV block")**
Ventricular rate is a fraction of the atrial rate, for example:
2:1 block = 150 bpm
3:1 block = 100 bpm
4:1 block = 75 bpm

**Atrial flutter with a 3:1 block**



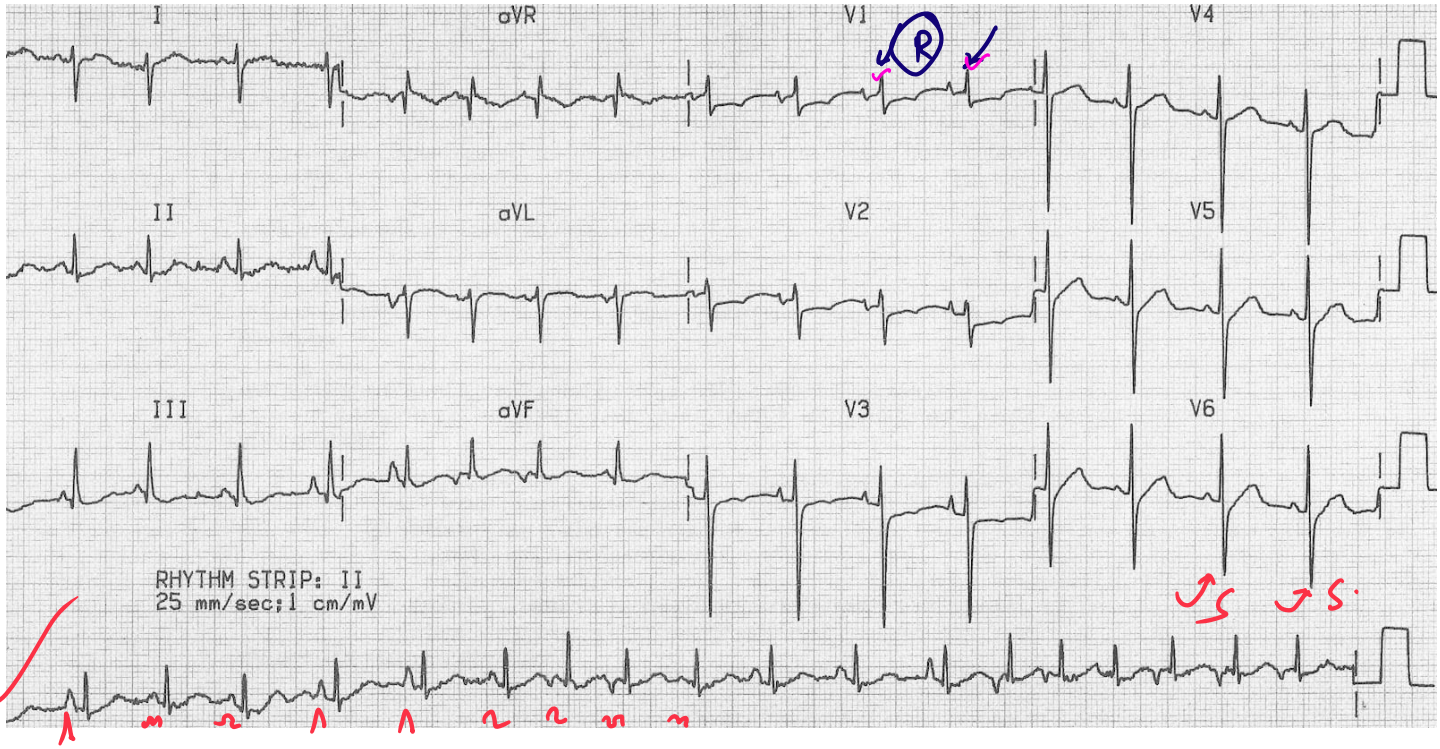

Inverted flutter waves in II, III + aVF at a rate of 300 bpm (one per big square)
Upright flutter waves in V1 simulating P waves
2:1 AV block resulting in a ventricular rate of 150 bpm
Note the occasional irregularity, with a 3:1 cycle seen in V1-3

# Multifocal Atrial Tachycardia (MAT)

A **rapid, irregular atrial rhythm arising from multiple ectopic foci** within the atria.
Most commonly seen in patients with severe COPD or congestive heart failure.
It is typically a **transitional rhythm between frequent premature atrial complexes (PACs) and atrial flutter / fibrillation.**

Heart rate **> 100 bpm** (usually 100-150 bpm; may be as high as 250 bpm).
**Irregularly irregular rhythm with varying PP, PR and RR intervals.**
At **least 3 distinct P-wave morphologies** in the same lead.
**Isoelectric baseline between P-waves (i.e. no flutter waves).**
**Absence of a single dominant atrial pacemaker** (i.e. not just sinus rhythm with frequent PACs).
**Some P waves may be nonconducted;** others may be aberrantly conducted to the ventricles.

Rapid irregular rhythm > 100 bpm.
At least 3 distinctive P-wave morphologies (arrows)

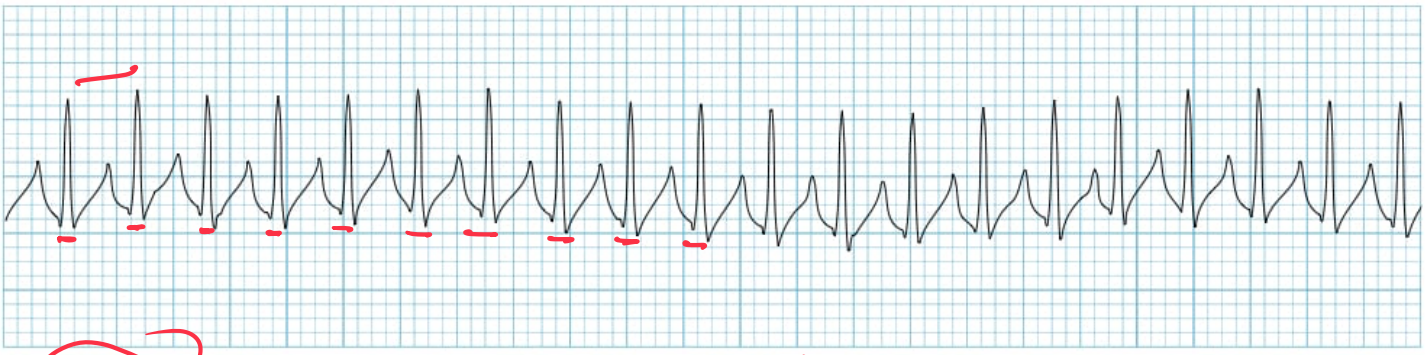



Rapid, irregular rhythm with multiple P-wave morphologies (best seen in the rhythm strip).
Right axis deviation, **dominant R wave in V1 and deep S wave in V6** suggest right ventricular hypertrophy due to cor pulmonale.

# SVT

any tachydysrhythmia arising from **above the level of the Bundle of His, and encompasses regular atrial, irregular atrial, and regular atrioventricular tachycardias**

In the **absence of aberrant conduction** (e.g. bundle branch block), **the ECG will demonstrate a narrow complex tachycardia**



(**SVT**): Rhythm strip demonstrating a regular, narrow-complex tachycardia

**Paroxysmal SVT (pSVT)** describes an SVT with abrupt onset and offset – characteristically seen with re-entrant tachycardias involving the AV node such as AVNRT or atrioventricular re-entry tachycardia (AVRT)

## Classification

SVTs can be classified based on:
**Site of origin (atria or AV node)** or;
**Regularity (regular or irregular)**
Classification based on QRS width is unhelpful as this is also influenced by the presence of pre-existing bundle branch block, rate-related aberrant conduction, or presence of accessory pathways.

**Classification of SVT by site of origin and regularity**

**Regular Atrial**

- Sinus tachycardia
- Atrial tachycardia
- Atrial flutter
- Inappropriate sinus tachycardia
- Sinus node reentrant tachycardia

**Irregular Atrial**

- Atrial fibrillation
- Atrial flutter (variable block)
- Multifocal atrial tachycardia

MAT

**Regular Atrioventricular**

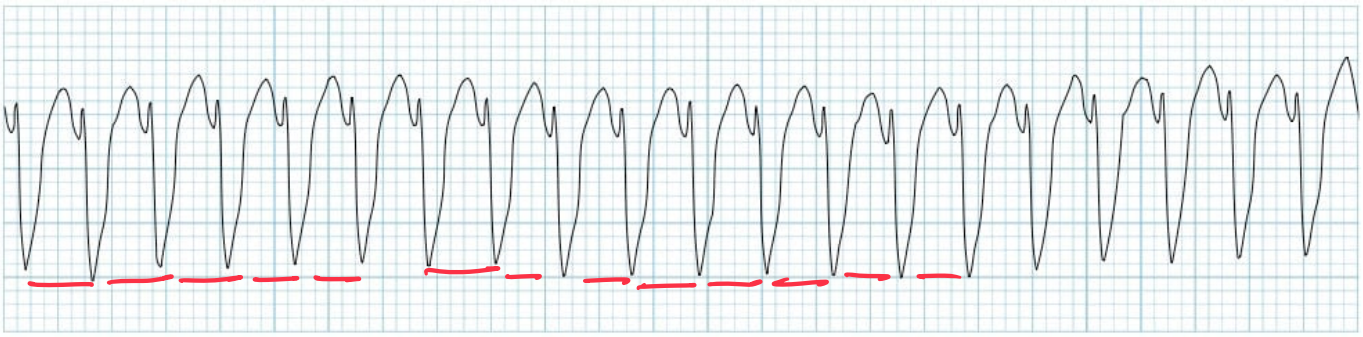
- AVRT
- AVNRT
- Automatic junctional tachycardia

**Ventricular Tachycardia (VT) is** a broad complex tachycardia originating from the ventricles. There are several different forms of VT — the most common is **monomorphic VT, which originates from a single focus within the ventricles.**

## ECG features of monomorphic VT

**Regular**, broad complex tachycardia

**Uniform QRS complexes within each lead — each QRS is identical (except for fusion/capture beats)**



Monomorphic VT: Regular, broad complex tachycardia

Other ECG features suggestive of VT include:

**Very broad complexes (>160ms)** >4 small boxes.

Absence of typical RBBB or LBBB morphology

**Extreme axis deviation ("northwest axis")**

**AV dissociation (P and QRS complexes at different rates)**

**Capture beats** — occur when the sinoatrial node transiently 'captures' the ventricles, in the midst of AV dissociation, to produce a **QRS complex of normal duration**

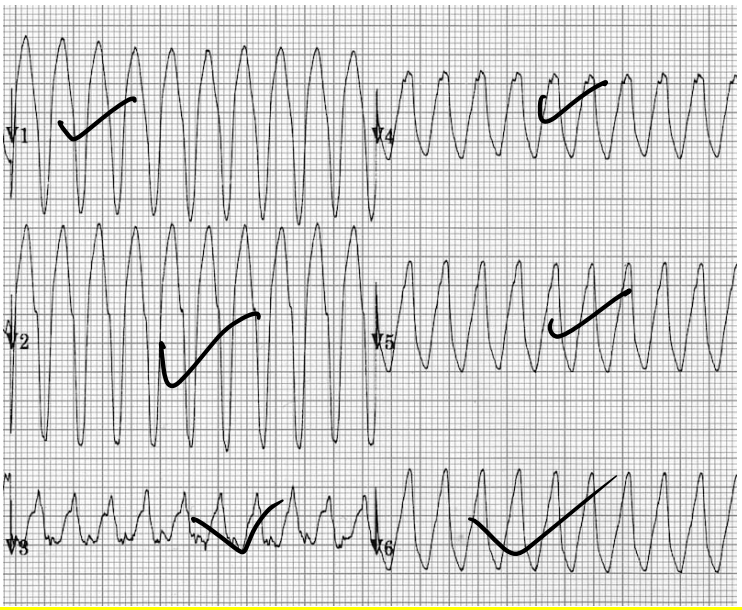
***Fusion beats*** — occur when a sinus and ventricular beat coincide to produce a hybrid complex of intermediate morphology

**Positive or negative concordance throughout the chest leads**, i.e. leads V1-6 show entirely positive (R) or entirely negative (QS) complexes, with no RS complexes seen

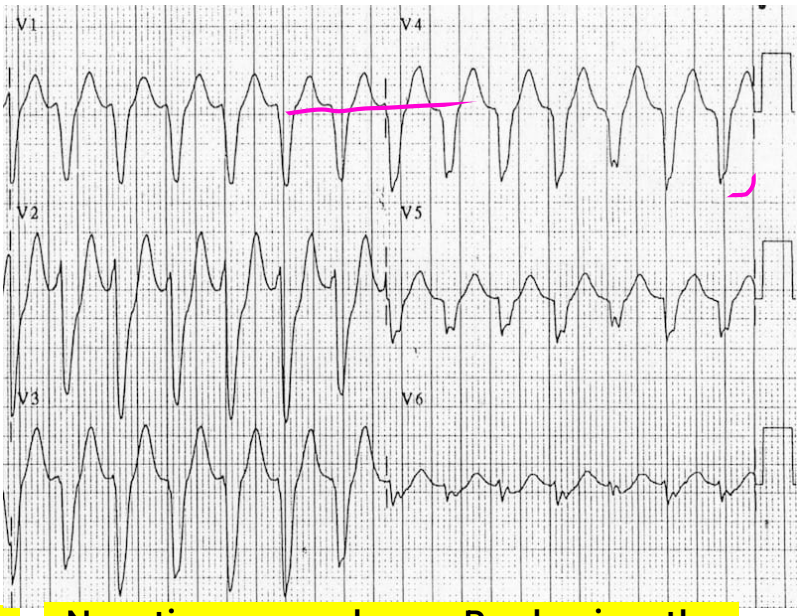
**Josephson's sign – Notching near the nadir of the S-wave**

**Brugada's sign** – The distance from the onset of the QRS complex to the nadir of the S-wave is >100ms

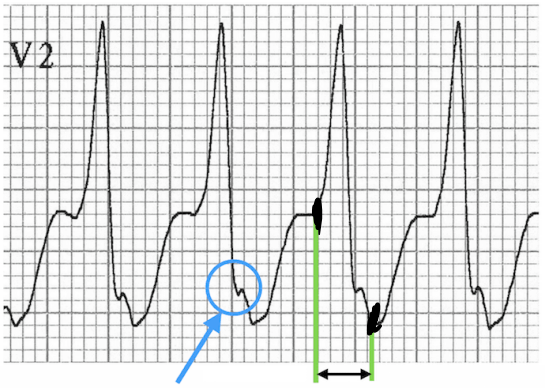
**RSR' complexes with a taller "left rabbit ear". This is the most specific finding in favour of VT. This is in contrast to RBBB, where the right rabbit ear is taller**




**Positive concordance: Predominantly positive QRS complexes throughout precordial leads**

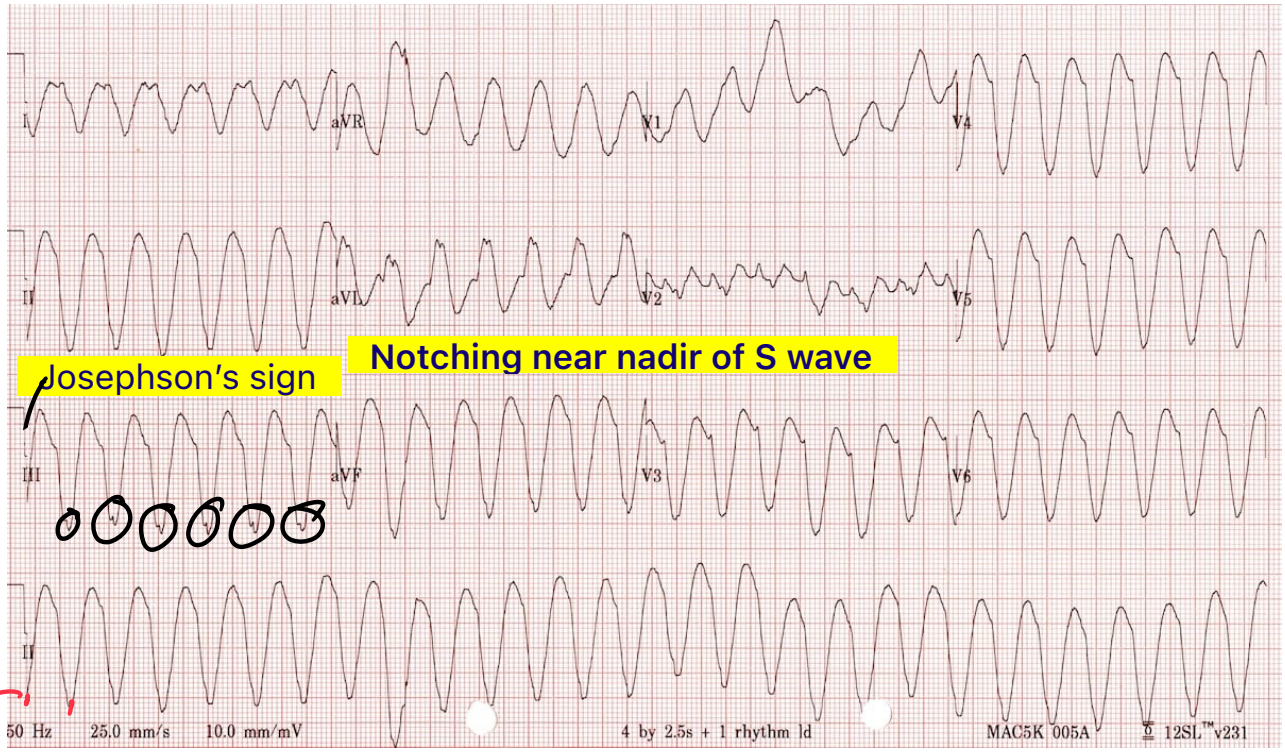



**Negative concordance: Predominantly negative QRS complexes throughout precordial leads**




Josephson sign

V1 V2 V3 → +
V4 V5 V6 — 0
V1 — V6 + −



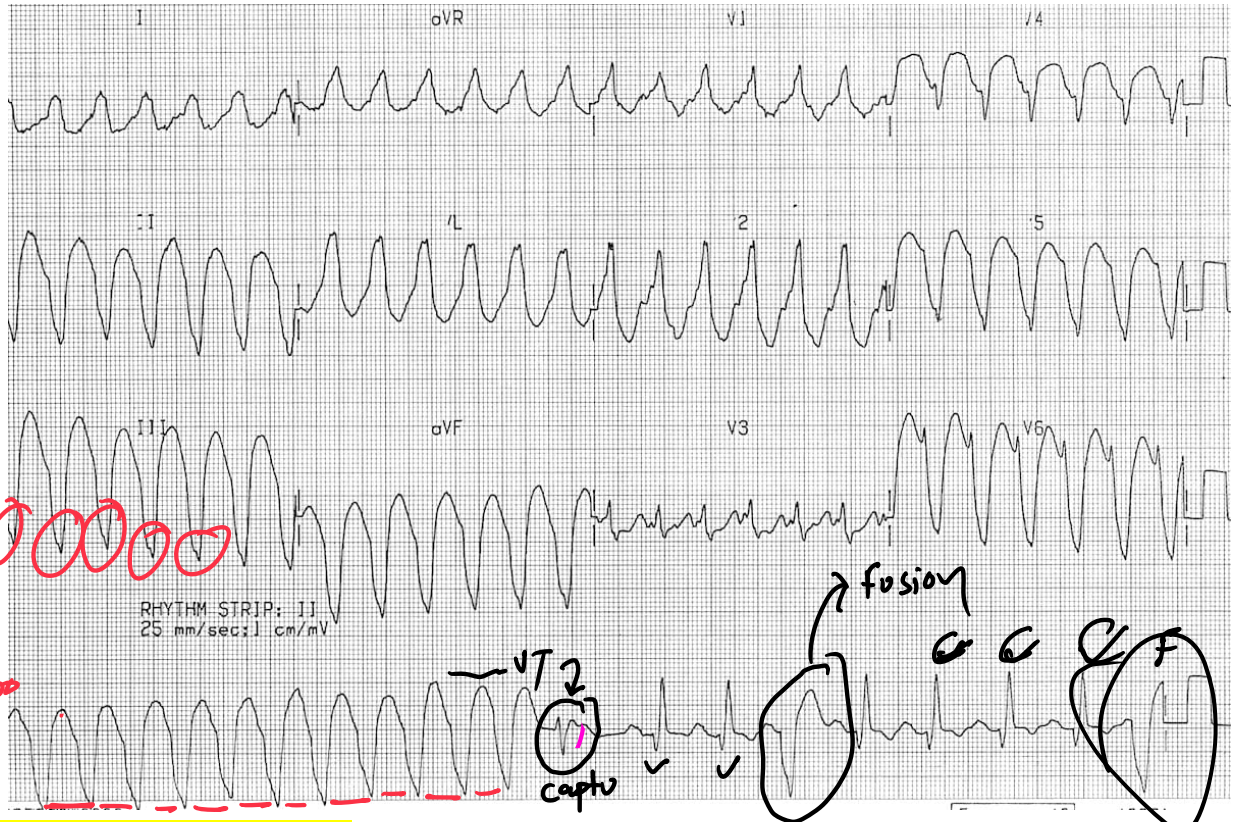

Josephson's sign

Notching near nadir of S wave

9×4 = 360

## Monomorphic VT:

Classic monomorphic VT with **uniform QRS complexes Indeterminate axis**
**Very broad QRS (~200 ms)**
**Notching near the nadir of the S wave in lead III = Josephson's sign**

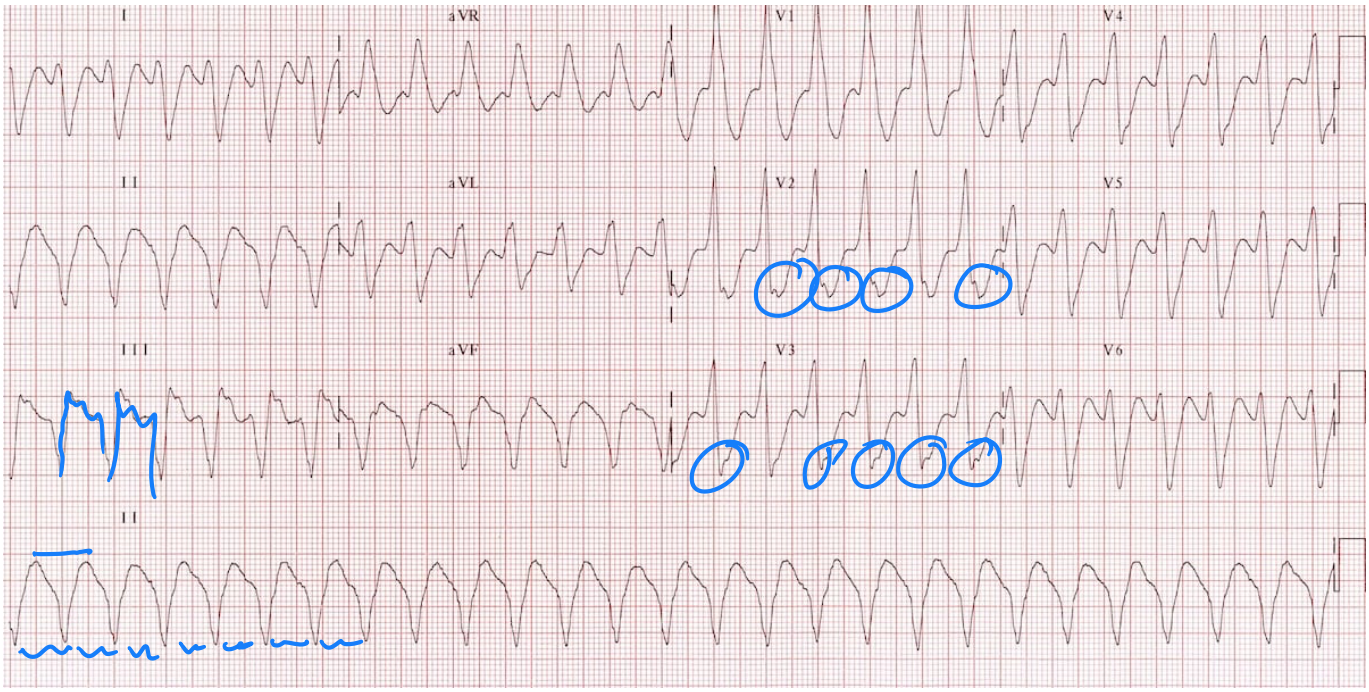



400

## Monomorphic VT:

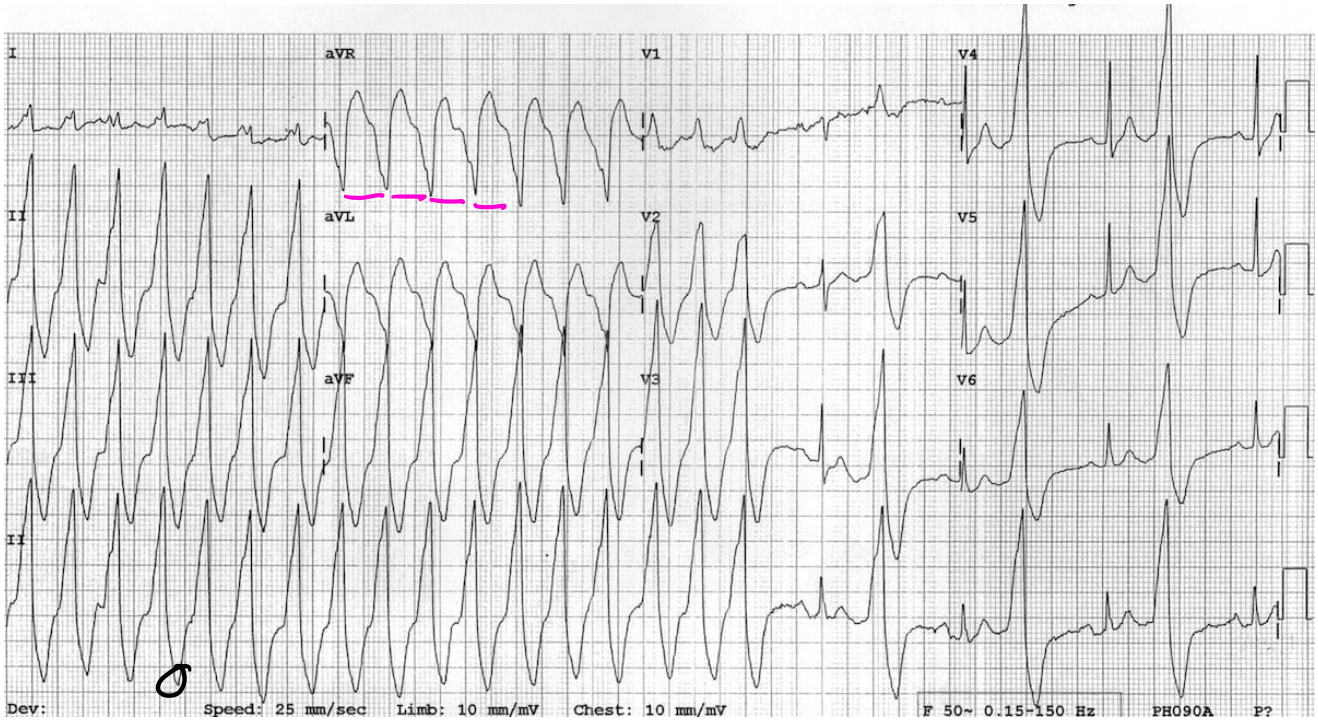
**Very broad QRS** complexes (~ 200 ms) with uniform morphology
**Fusion and capture beats** are seen in the rhythm strip
**Brugada's sign is present**: the time from the onset of the QRS complex to nadir of S wave is > 100 ms (best seen in V6)

7.5 small boxes



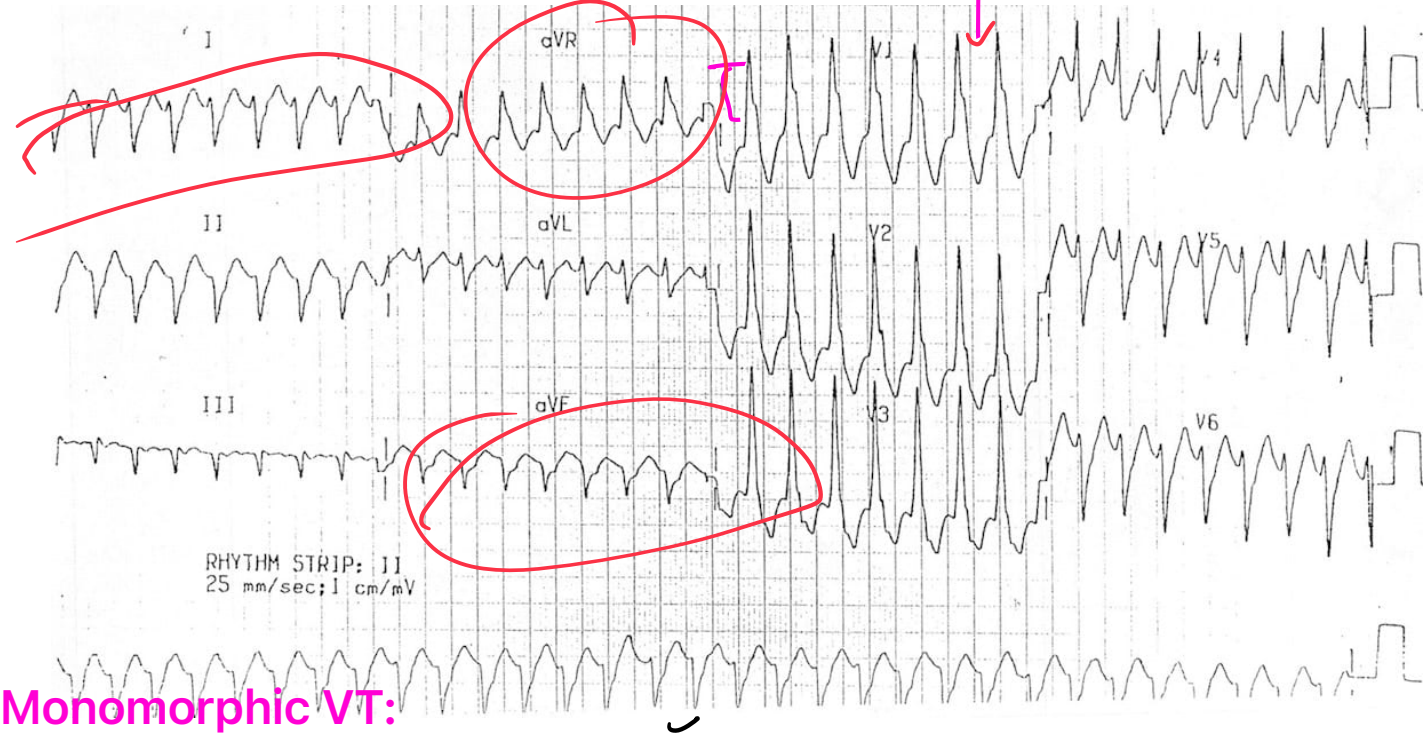

## Monomorphic VT:

**Very broad complexes (~ 200 ms in V5-6)**
**Northwest axis (-120 degrees)**
**Brugada's sign — the distance from the onset of the QRS complex to the nadir of the S-wave is > 100ms**
**Josephson's sign — notching near the nadir of the S wave is seen in leads II, III, aVF**
**Possibly some superimposed P waves in aVF**




**Monomorphic VT alternating with ventricular bigeminy.**

**The ventricular complexes have the following features:**
**Very broad QRS** duration (> 160 bpm)
**Positive concordance in the precordial leads** (dominant R waves in V1-6)
**Brugada's sign** – time from onset of QRS to nadir of S wave > 100 ms; best seen in leads aVR and aVL




## Monomorphic VT:

Extreme axis deviation / northwest axis is present

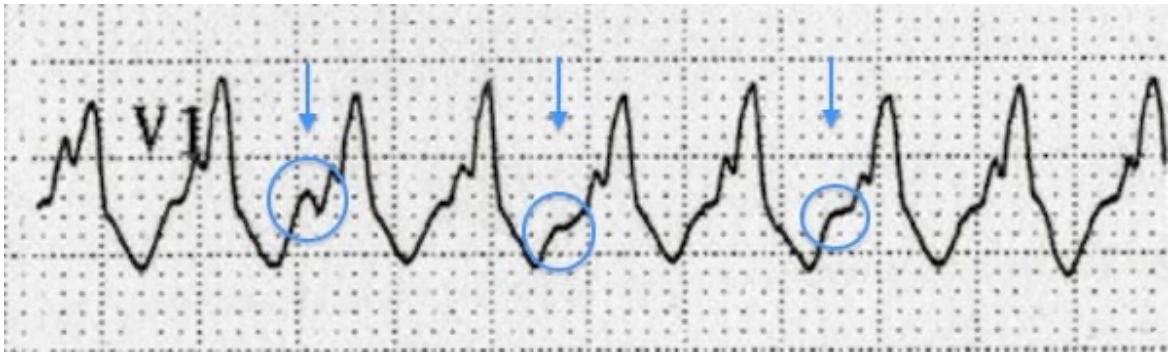
-150 degree; QRS positive in aVR, negative in I + aVF

There is a RBBB-like pattern in V1 with a taller left rabbit ear – this is very specific for VT

## Monomorphic VT:

Northwest axis

Tall monophasic R wave in V1 with an rS complex in V6 (small R wave, big S wave) — this pattern is also very specific for VT

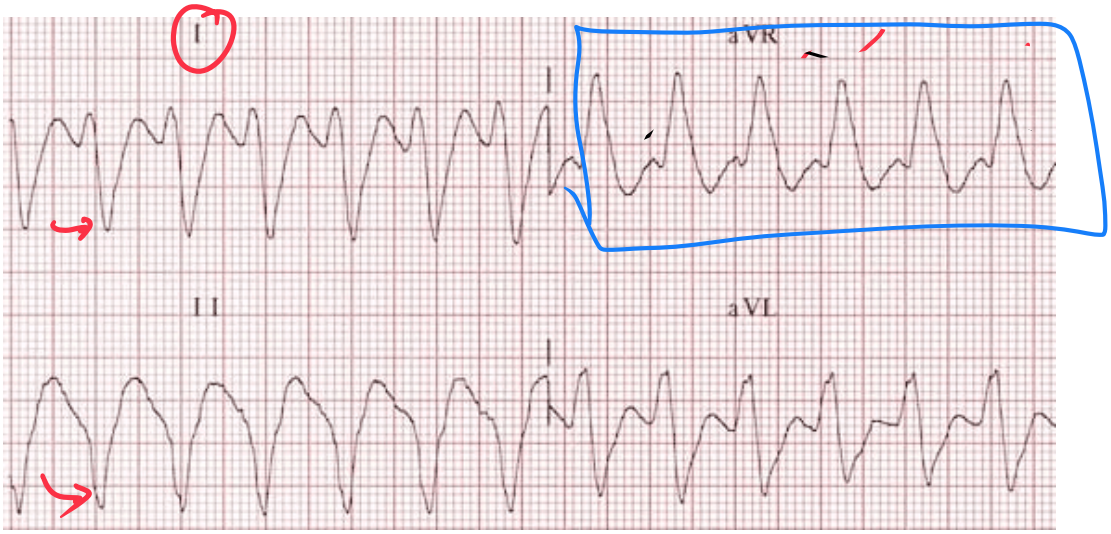



AV dissociation: superimposed P waves at a different rate to the QRS complexes

# VT VS SVT

## #1: A is for Axis

Is there a Northwest axis?
Presence of a **Northwest axis (-90 to -180 degrees) is highly specific for VT (90%).**
**Look for a dominant R wave in aVR for quick reference**. Always consider metabolic disturbance (K, Na channel blockade, acidosis).




**Northwest axis: Dominant R wave in aVR, dominant S wave in I and II**

## #2: B is for Broad complexes

40x5 = 200

Use a **cut-off of > 200ms**, which has a **specificity of 85-90% for VT**. Below this, there is too much overlap between VT and SVT with aberrancy.

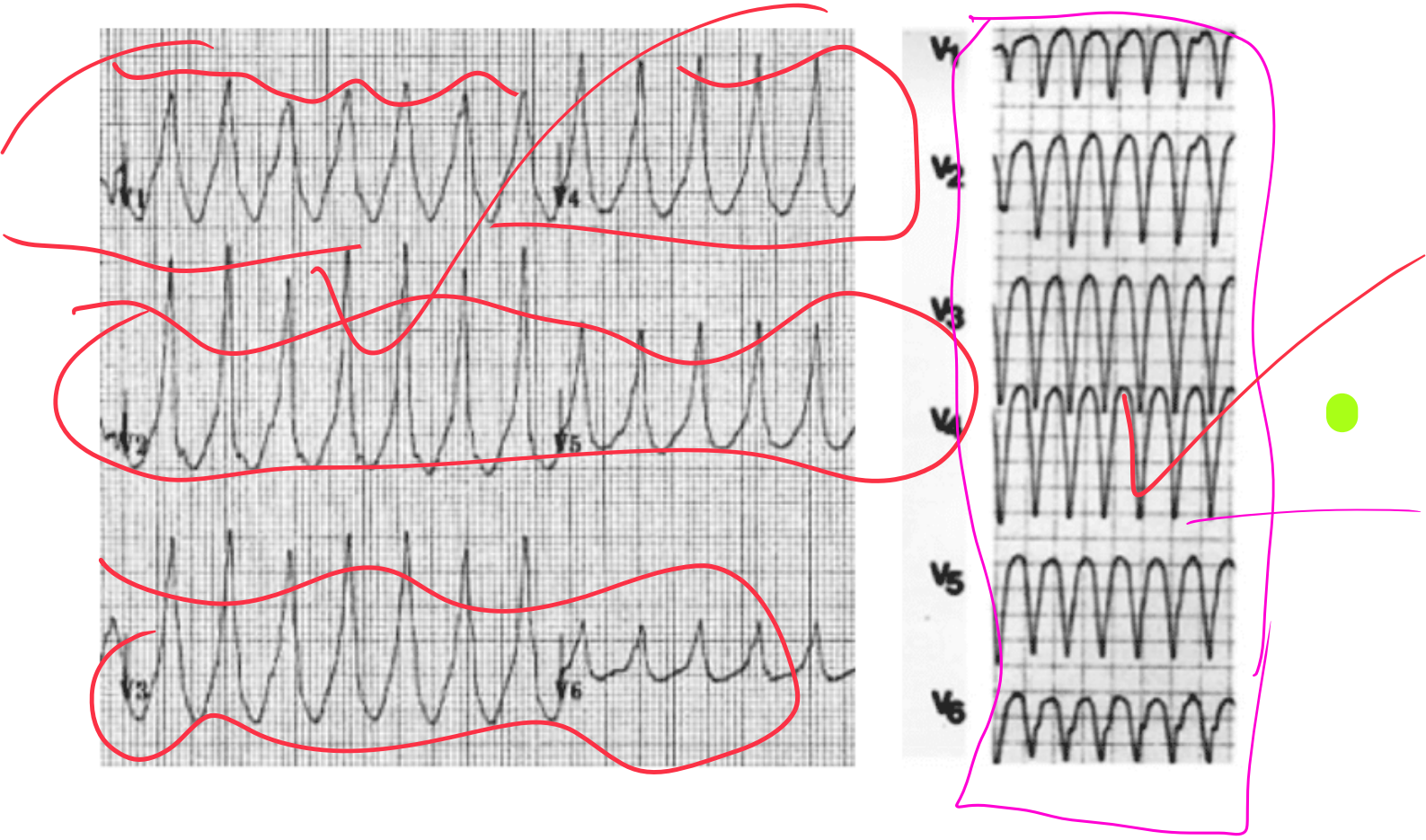
Note that variants such as Right Ventricular Outflow Tract (RVOT) VT may demonstrate shorter QRS durations – as such, a narrower QRS does not exclude VT.

## #3: C is for Concordance

**If you gave me just one question, this would be it – does the QRS complex resemble any form of aberration?**

**If so, SVT is probable**
**If not, VT or antidromic AVRT is probable**
**Precordial concordance (a lack of RS complex in V1-6) is 97% specific for VT.**

**Positive concordance (left) refers to entirely positive complexes in lead V1-6, with no rS complexes seen. Vice versa, negative concordance (right) is entirely negative (QS) complexes in V1-6.**

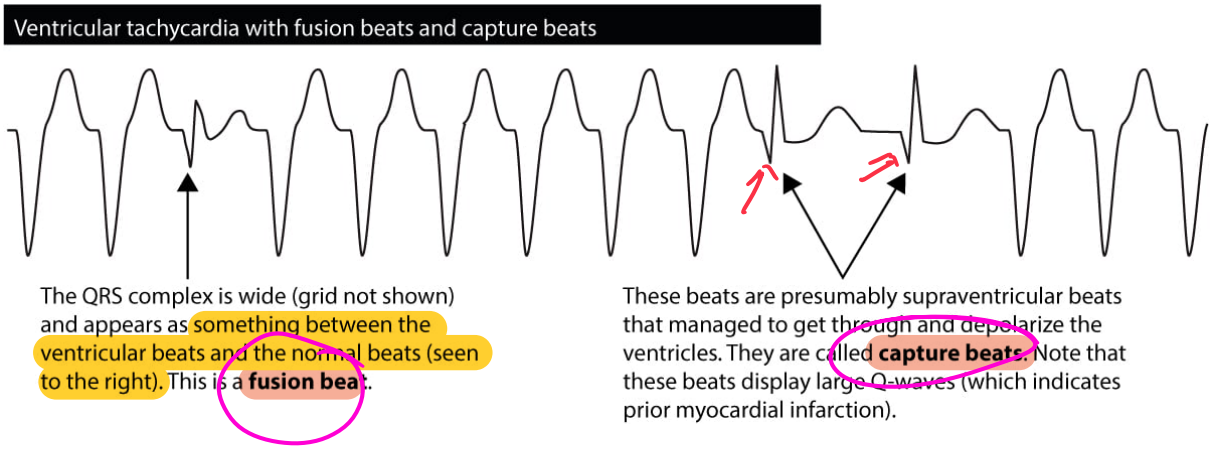



Positive concordance (left): Entirely positive complexes in V1-6
Negative concordance (right): Entirely negative (QS) complexes in V1-6

## #4: D is for Dissociation

Is there AV dissociation?
Any evidence of AV dissociation is highly specific for VT:

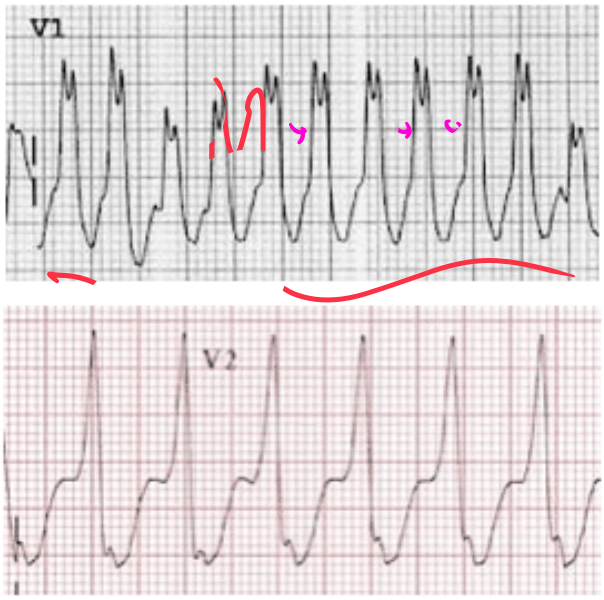
P and QRS complexes at different rates
Capture beats: where the SA node transiently "captures" the ventricles, producing an isolated normal QRS complex
Fusion beats: Occur when a sinus and ventricular beat coincide to produce a hybrid complex (see Dressler beat)



# #5: E is for Early part of QRS

Is the early part of the QRS fast or slow?
SVT with aberrancy and VT differ in the way that they engage the His-Purkinje network:
VT propagates from ventricular muscle, with initial slow myocyte-to-myocyte conduction time. This causes a delayed/slurred initial QRS component

SVT with aberrancy displays initial sharp QRS deflections that arise from the preserved bundle branch




**SVT with aberrancy**

Note rapid upstroke of R wave, < 40ms duration

**VT** → Myocytes → slow

Time to peak of depolarisation (R wave) is > 40ms

In VT, time to peak of depolarisation is often delayed at > 40ms in V1 or V2

## The VT score

- Dominant R wave in aVR
- No RS complexes in V1-6
- Lead II RWPT > 50ms
- AV dissociation
- Notched S wave in V1
- Dominant R wave in V1
- Initial R > 40ms in V1 or V2

Score of 0: 15% specific
Score of 1: 55%
Score of 2: 78%
Score of 3: 99.6%
Score of 4: 100%

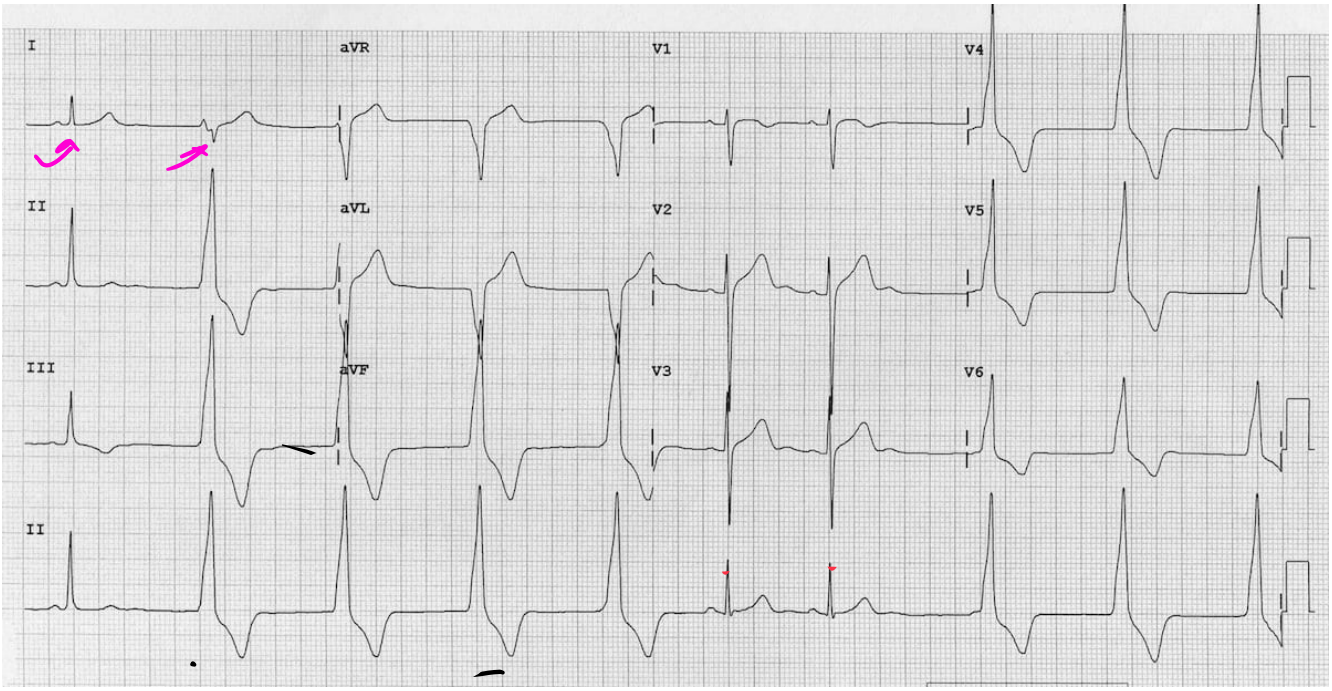
slow

# Accelerated Idioventricular Rhythm (AIVR) Overview

rate of an ectopic ventricular pacemaker exceeds that of the sinus node.
Often associated with increased vagal tone and decreased sympathetic tone

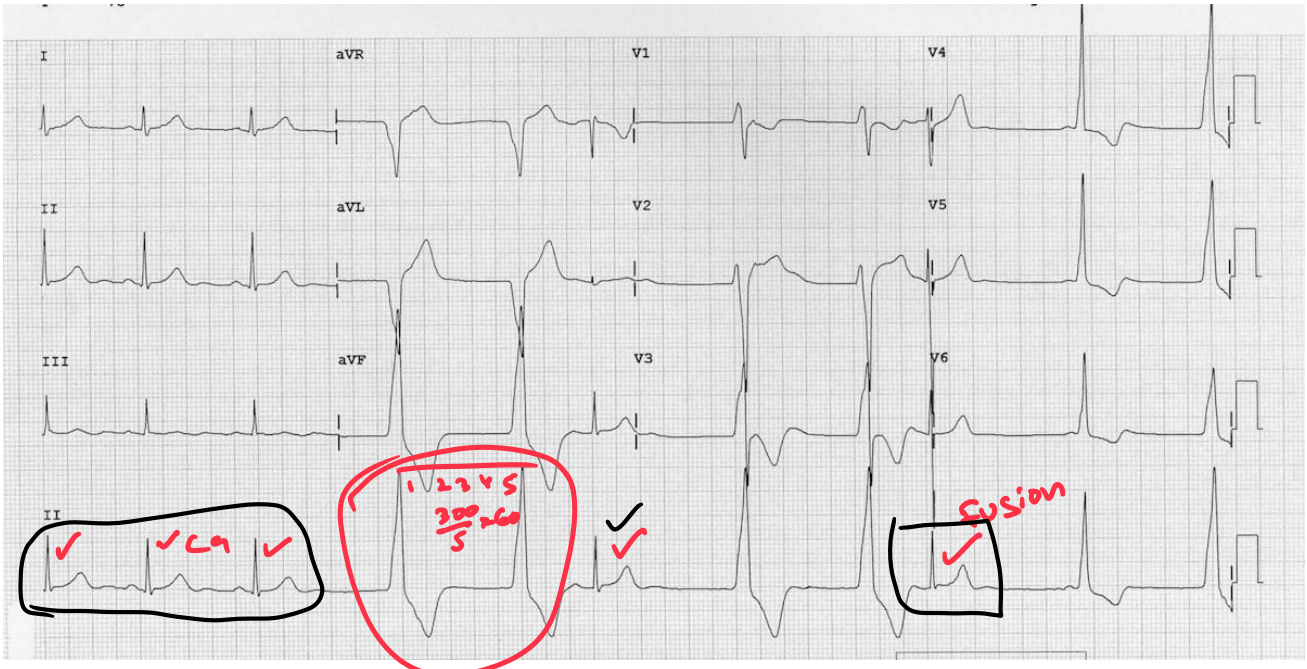
- Regular rhythm
- Rate typically 50-120 bpm
- Three or more ventricular complexes; QRS duration > 120ms
- Fusion and capture beats

AIVR is classically seen in the reperfusion phase of an acute STEMI, e.g. post thrombolysis
Usually a well-tolerated, benign, self-limiting arrhythmia




Ventricular rhythm at 60 bpm
Multiple sinus capture beats
Competing sinus and idioventricular pacemakers are present. There is an underlying sinus arrhythmia, with sinus capture occurring when the sinus rate exceeds the idioventricular rate.

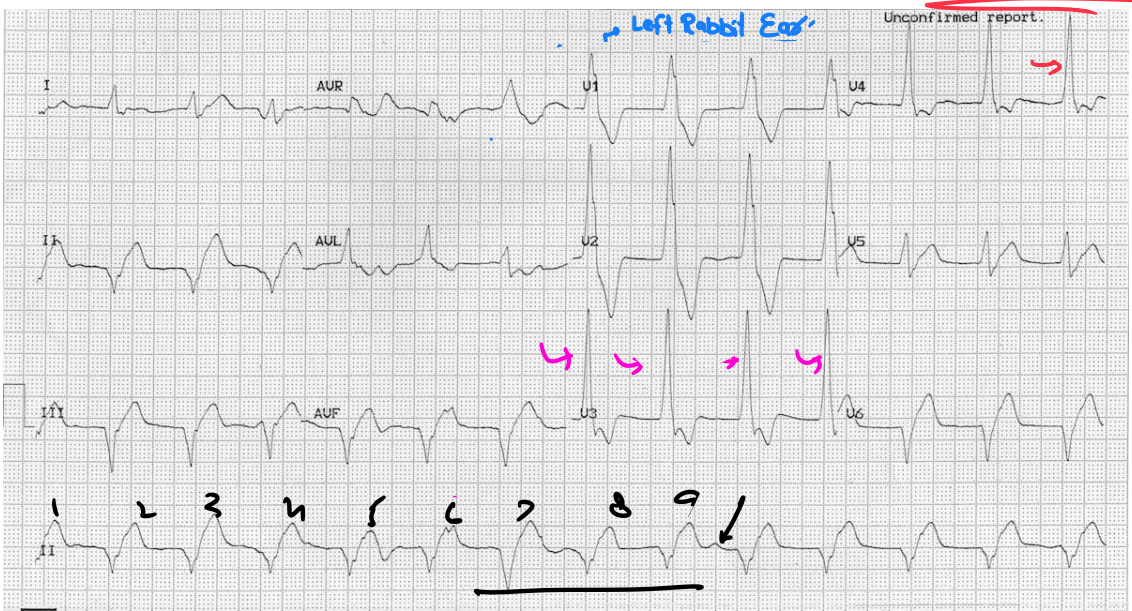
This patient was a healthy 36-year old marathon runner with presumably very high resting vagal tone causing sinus bradycardia and sinus arrhythmia.



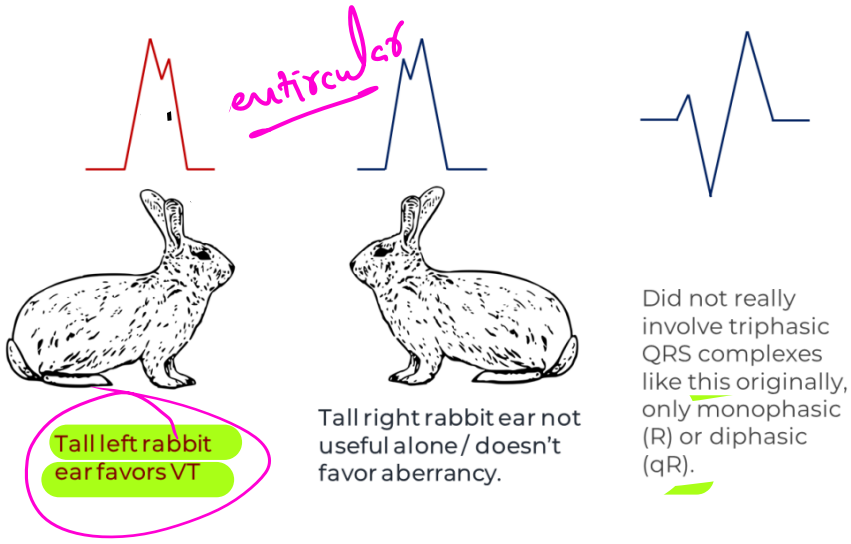

Another ECG from the same patient showing:

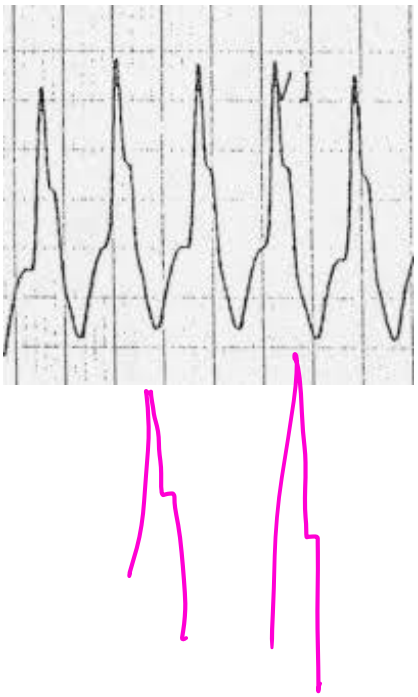
AIVR at 60 bpm
Isorhythmic AV dissociation with frequent sinus capture beats
A fusion beat

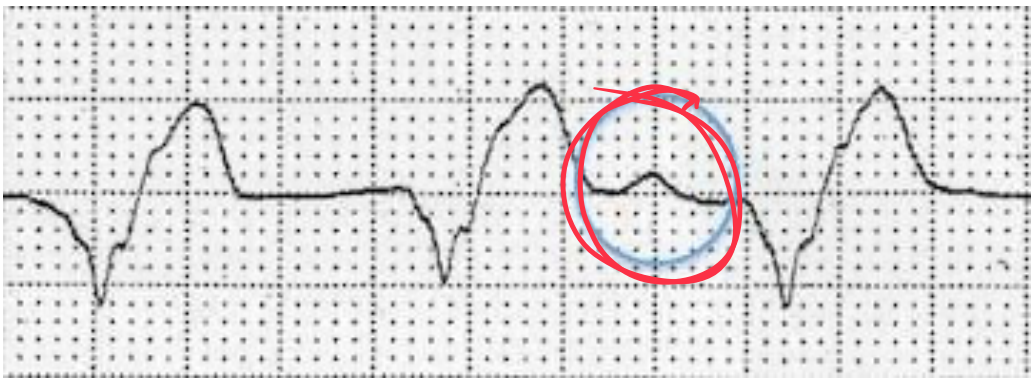
AIVR showing ventricular complexes (V), capture beat (C), fusion beat (F)



Ventricular rhythm at 75 bpm
AV dissociation — a dissociated P wave is seen in the rhythm strip (after 9th complex), another in lead aVL. Elsewhere, dissociated P waves cause intermittent deformation of the QRS complexes
The taller left rabbit ear sign is present — there is a notched R wave in V1 with a taller initial R wave; this is highly specific for a ventricular origin of the QRS complexes

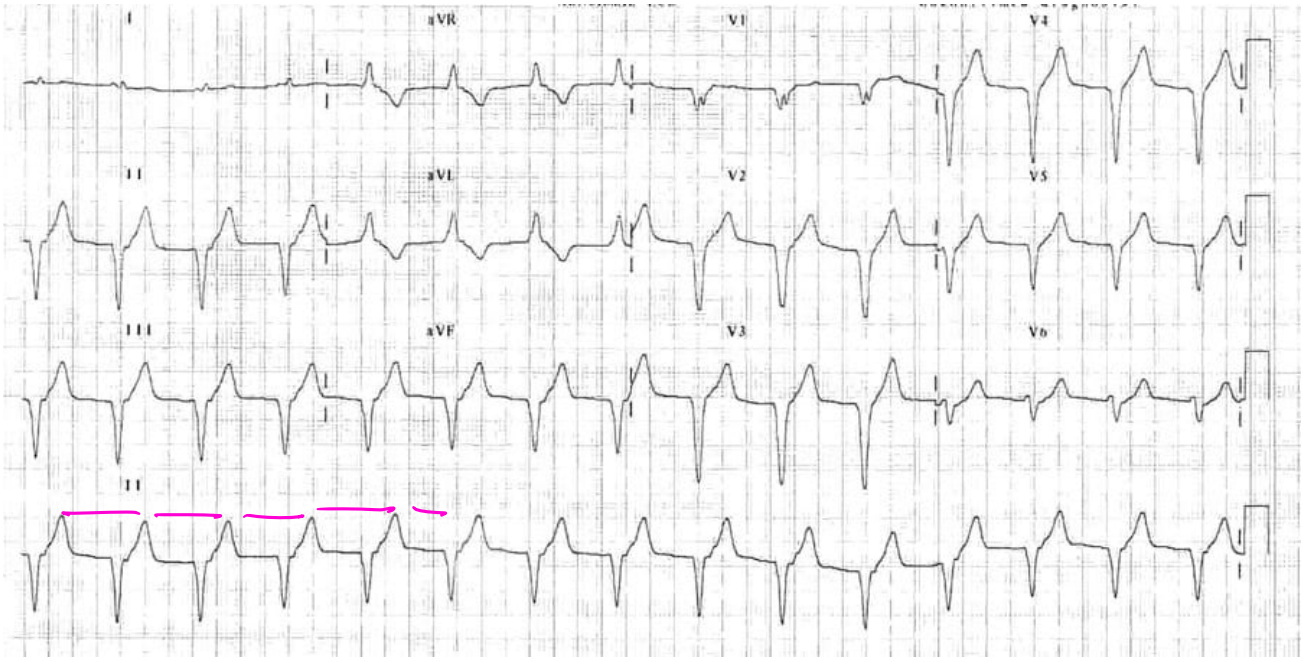


AIVR showing dissociated P waves (circled)

Taller left rabbit ear = ventricular origin of QRS complexes




Broad complex at 90 bpm
No visible P waves
This dysrhythmia occurred following reperfusion from an anterior STEMI.

# VT POINTS

Ventricular tachycardia with fusion beats and capture beats

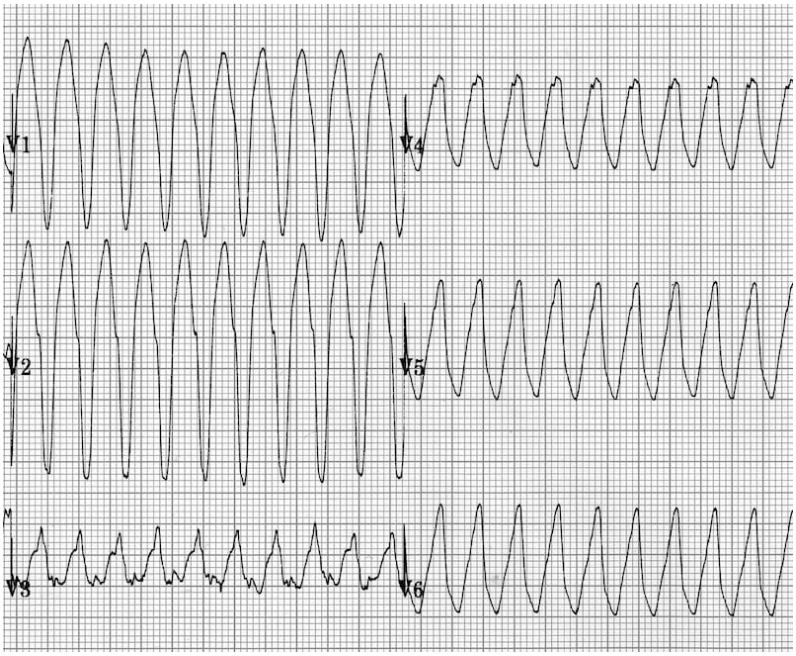
The QRS complex is wide (grid not shown) and appears as something between the ventricular beats and the normal beats (seen to the right). This is a **fusion beat**.

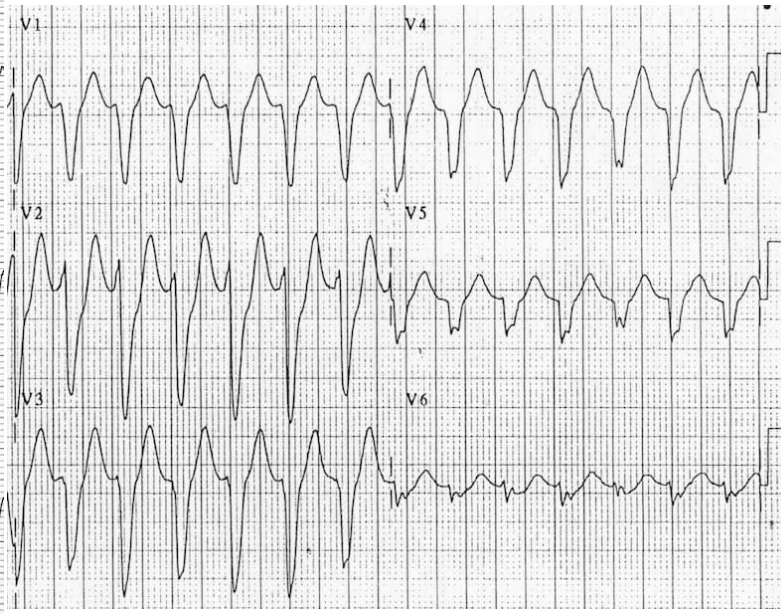
These beats are presumably supraventricular beats that managed to get through and depolarize the ventricles. They are called **capture beats**. Note that these beats display large Q-waves (which indicates prior myocardial infarction).

**Capture beats:** Occur when the sinoatrial node transiently "captures" the ventricle in the midst of AV dissociation, producing a QRS complex of normal duration

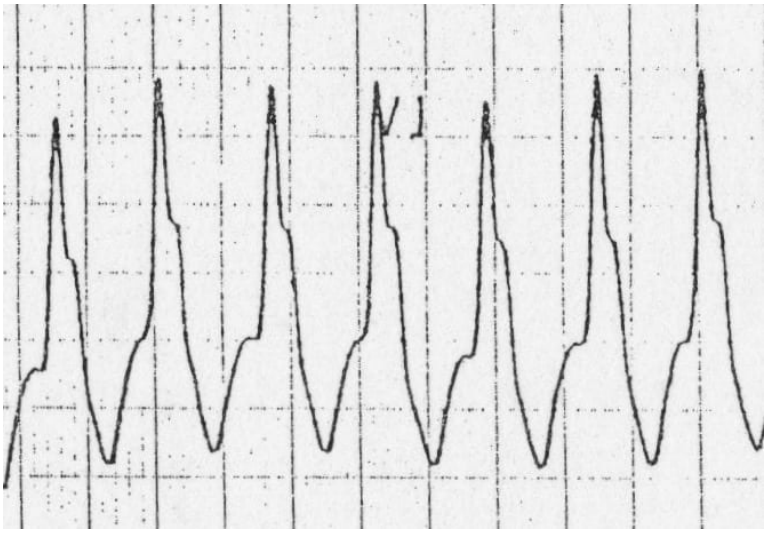
**Fusion beats:** Occur when a sinus and ventricular beat coincide to produce a hybric complex (see Dressler beat)

**Positive concordance throughout the precordial leads:**



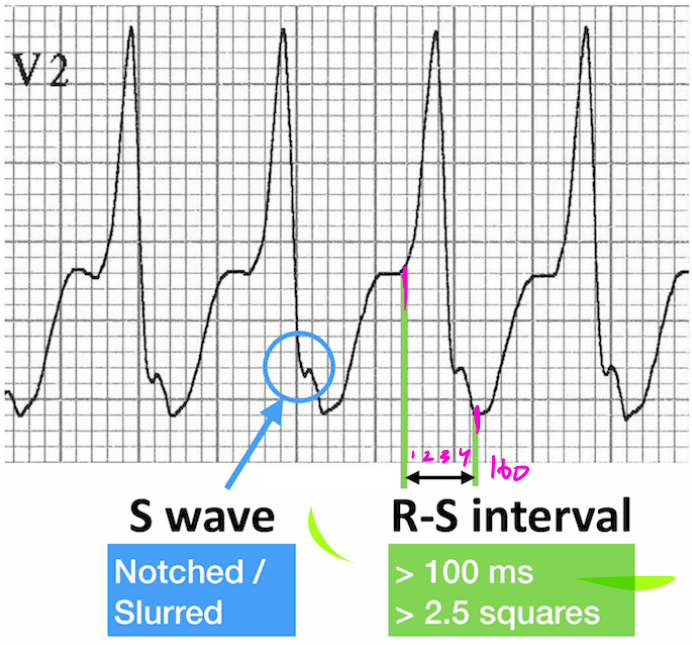




**Negative concordance in VT: Leads V1-6 show entirely negative (QS) complexes, with no rS complexes seen**




**RSR′ complexes with a taller left rabbit ear:**

**most specific finding in favour of VT**




**Josephson sign: Notching/ slurring near the nadir of the S wav**

**Brugada sign: Distance from onset of R wave to nadir of S wave is > 100ms in leads V1-6 (discussed further below)**

# PREMATURE VENTRICULAR COMPLEXES

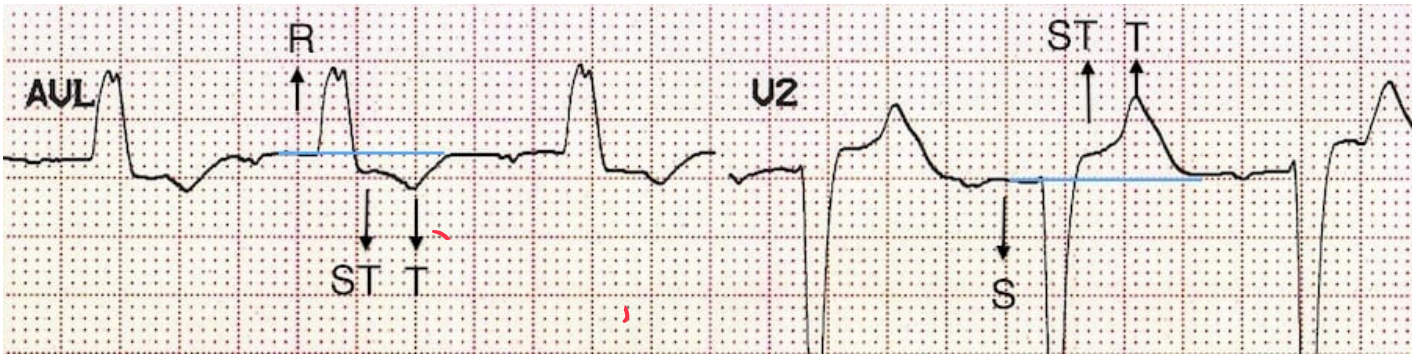
Broad QRS complex (≥ 120 ms) with abnormal morphology
Premature — i.e. occurs earlier than would be expected for the next sinus impulse
Discordant ST segment and T wave changes.
Usually followed by a full compensatory pause
Retrograde capture of the atria may or may not occur

Ectopic firing of a focus within the ventricles bypasses the His-Purkinje system and depolarises the ventricles directly
This disrupts the normal sequence of cardiac activation, leading to asynchronous activation of the two ventricles
The consequent interventricular conduction delay produces QRS complexes with prolonged duration and abnormal morphology

## Discordance

Appropriate discordance describes a pattern of repolarisation abnormality (typically seen with left bundle branch block, paced rhythms, VT) in which the ST segment and T wave are directed opposite to the main vector of the QRS complex. Because there is abnormal depolarisation, there is subsequent abnormal repolarisation which is discordant:

ST depression and T wave inversion in leads with a dominant R wave
ST elevation with upright T waves in leads with a dominant S wave



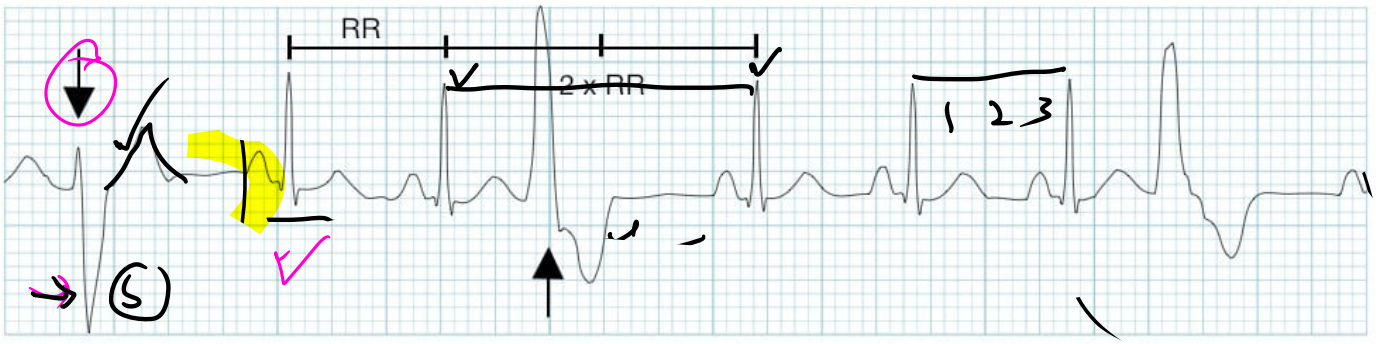

With a full compensatory pause, the next normal beat arrives after an interval that is equal to double the preceding R-R interval

**The origin of each PVC can be discerned from the QRS morphology:**

PVCs arising from the right ventricle have a left bundle branch block morphology (dominant S wave in V1)
PVCs arising from the left ventricle have a right bundle branch block morphology (dominant R wave in V1)

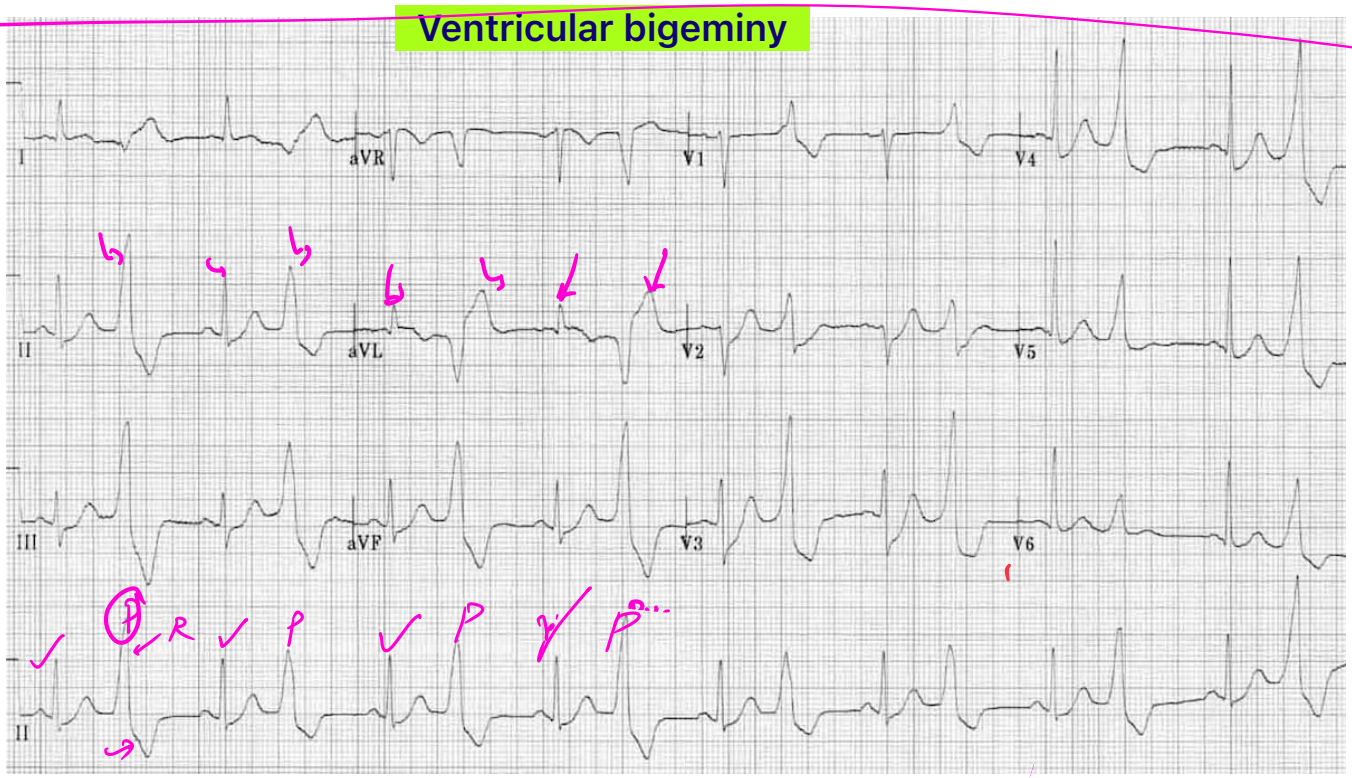
PVCs often occur in repeating patterns:

Bigeminy — every other beat is a PVC
Trigeminy — every third beat is a PVC
Quadrigeminy — every fourth beat is a PVC
Couplet — two consecutive PVCs
NSVT — between three and thirty consecutive PVCs (see below)

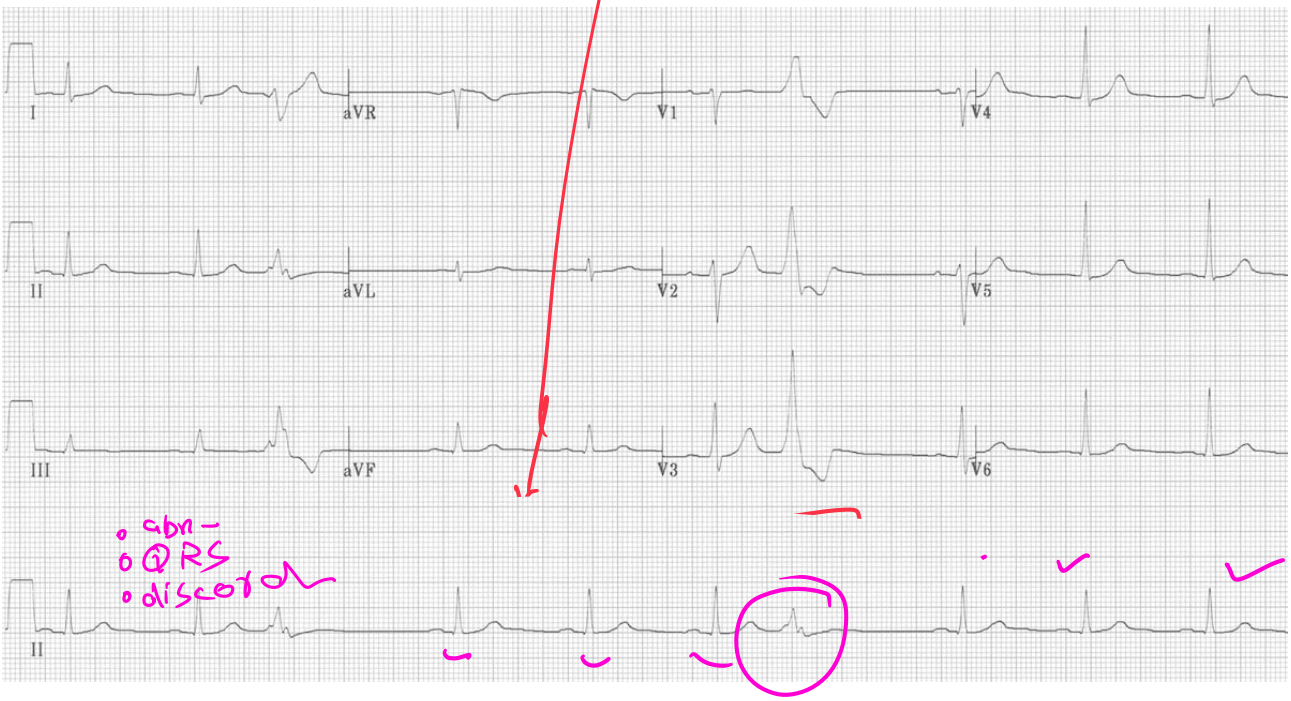



Sinus rhythm with PVCs of two different morphologies (arrows)
Note the appropriately discordant ST segments / T waves
The pause surrounding the PVC is equal to double the preceding R-R interval (= a full compensatory pause)

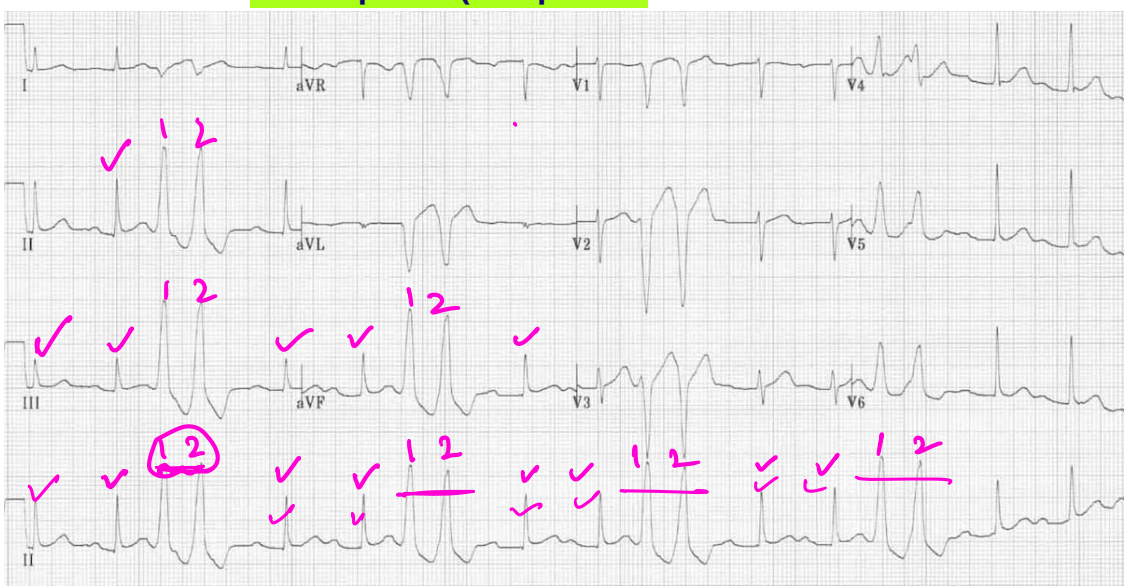
**Ventricular bigeminy**



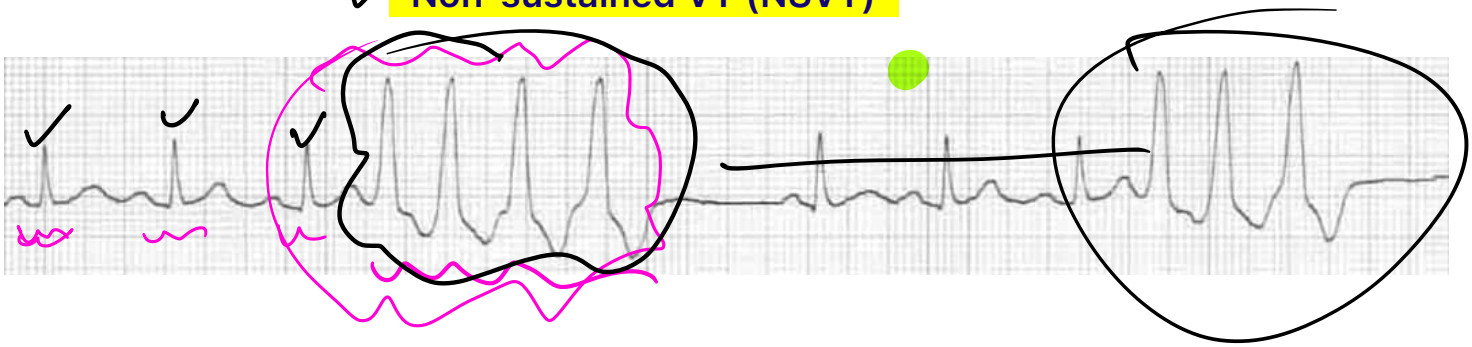
## Ventricular quadrigeminy



## PVC pairs (couplets



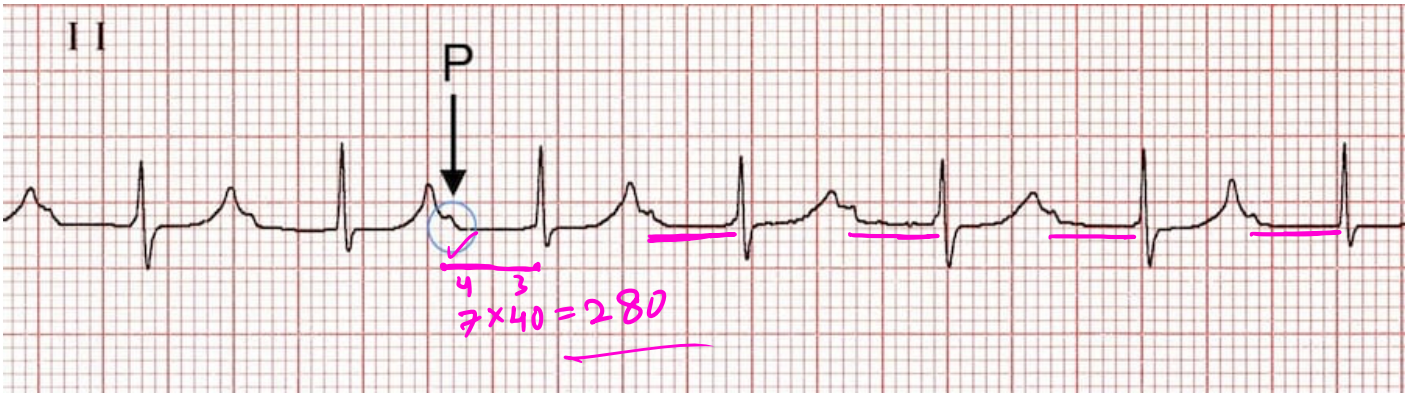
## Non-sustained VT (NSVT)



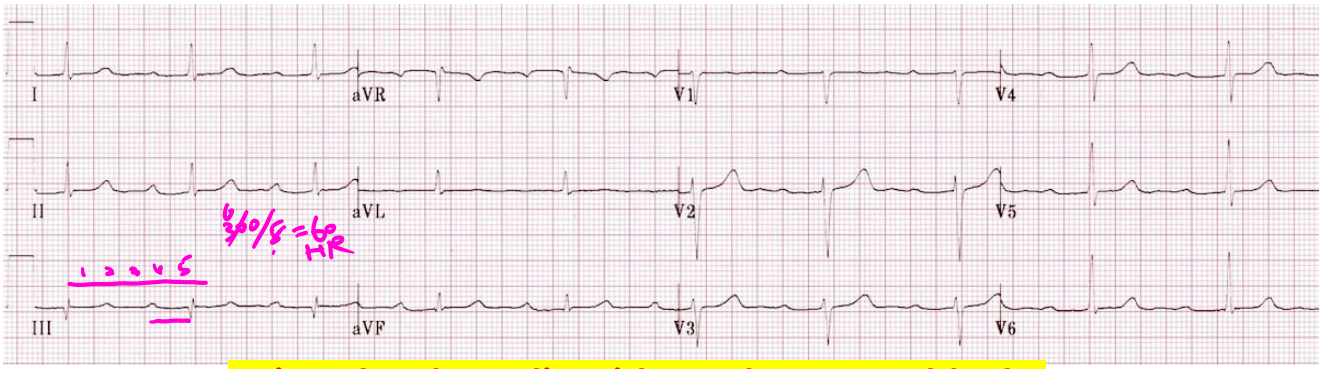
Definitions vary regarding 3 or more PVCs
Some authors define three PVCs as a triplet of PVCs; whilst others describe this as a 'short burst of VT'; but more commonly as NSVT
A consensus definition would be: 3-30 consecutive PVCs with a rate >100bpm described as non-sustained VT (ventricular rhythm if rate <100bpm)

## First Degree Heart Block

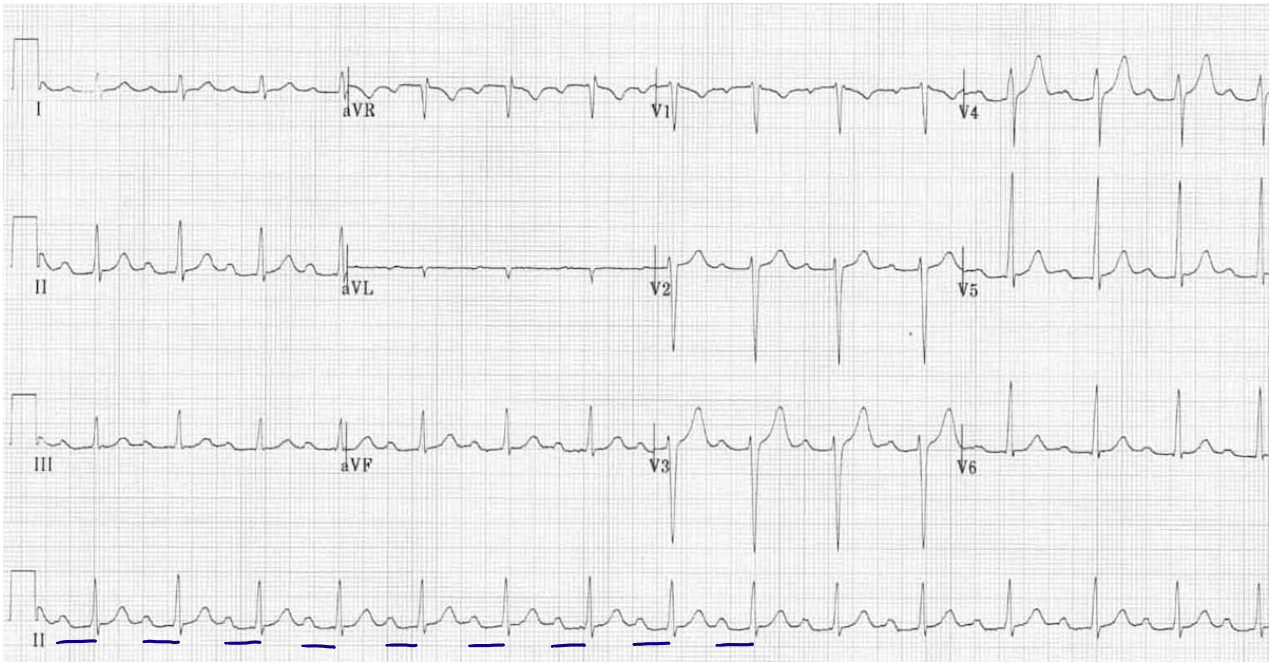
PR interval > 200ms (five small squares)




Marked first degree heart block
PR interval > 300 ms, P waves are buried in the preceding T wave



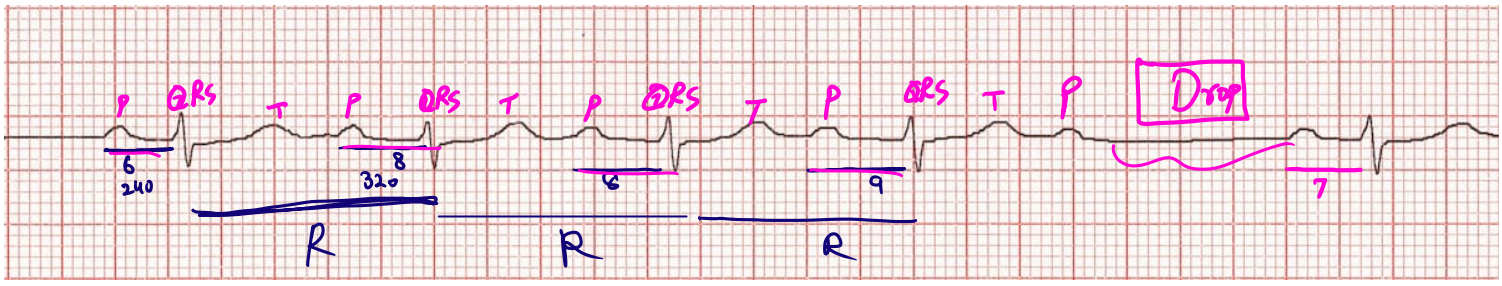

Sinus bradycardia with 1st degree AV block
PR interval > 300 ms




**Normal sinus rhythm with 1st degree AV block**
**PR interval 260 ms**

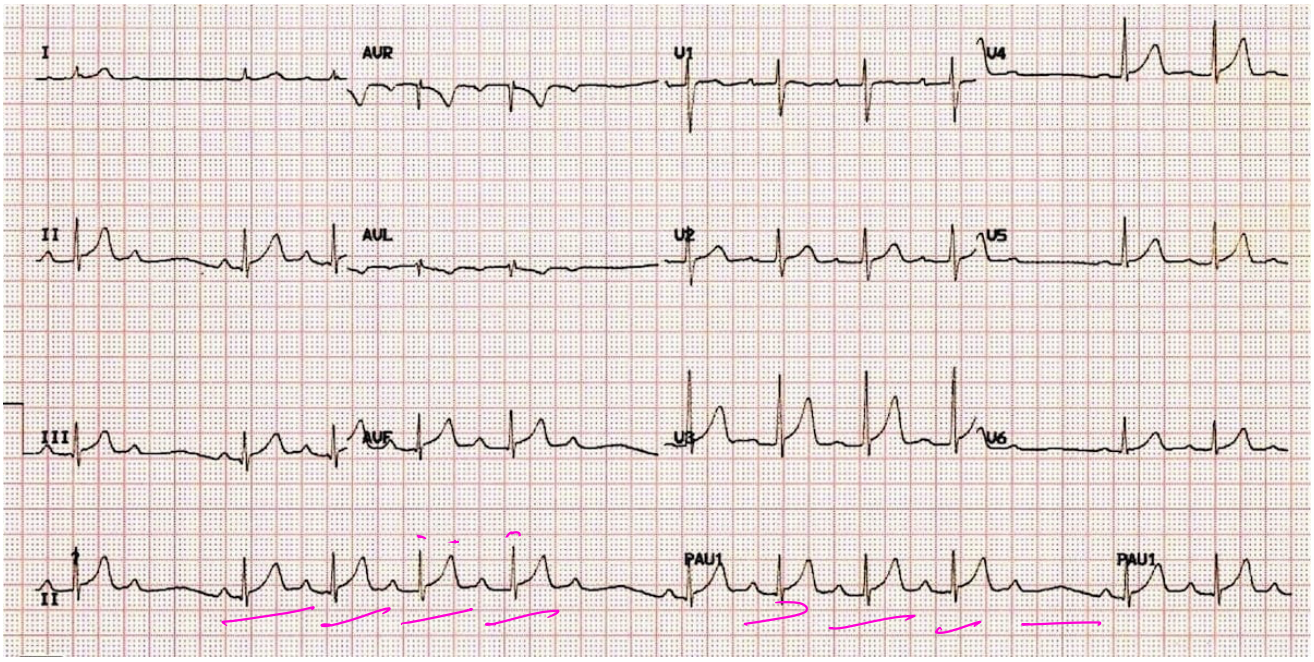
**Mobitz I block (Wenckebach phenomenon)**

**Progressive prolongation of the PR interval culminating in a non-conducted P wave:**
PR interval is longest immediately before dropped beat
PR interval is shortest immediately after dropped beat



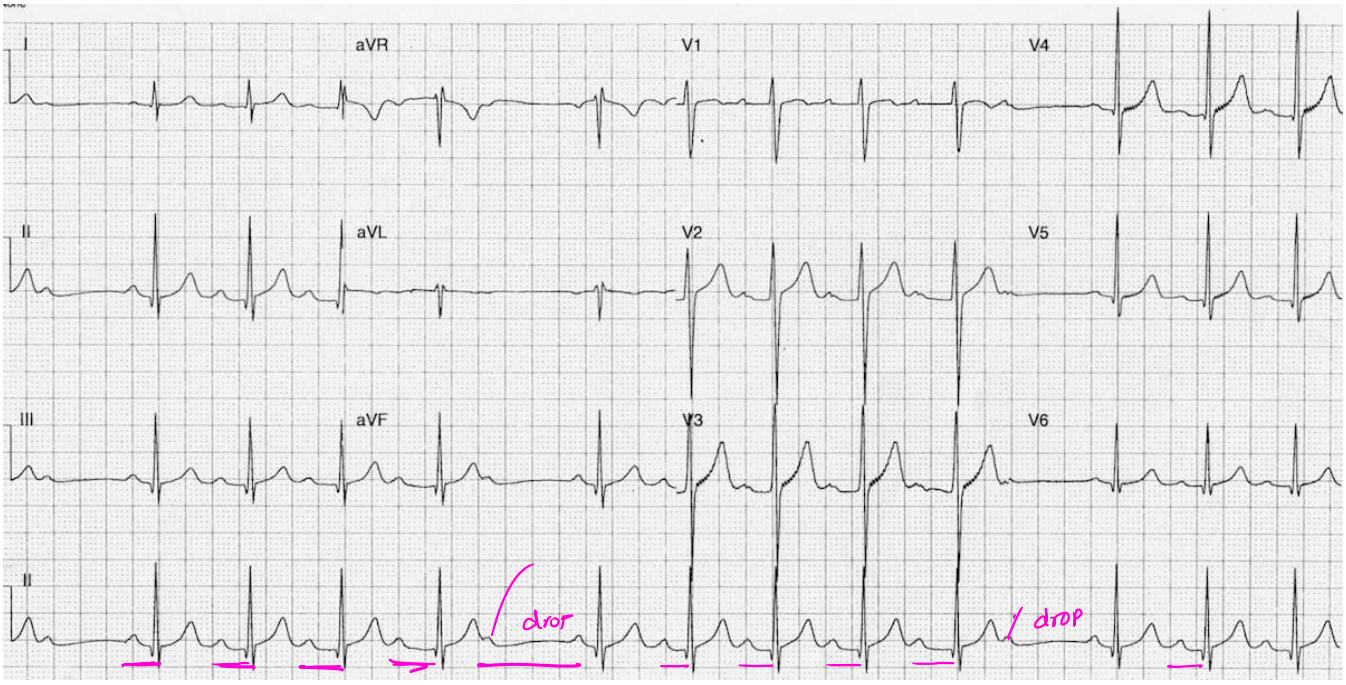

**AV block: 2nd degree, Mobitz type I**

The P-P interval remains relatively constant
The greatest increase in PR interval duration is typically between the first and second beats of the cycle
The RR interval progressively shortens with each beat of the cycle
The Wenckebach pattern tends to repeat in P:QRS groups with ratios of 3:2, 4:3 or 5:4



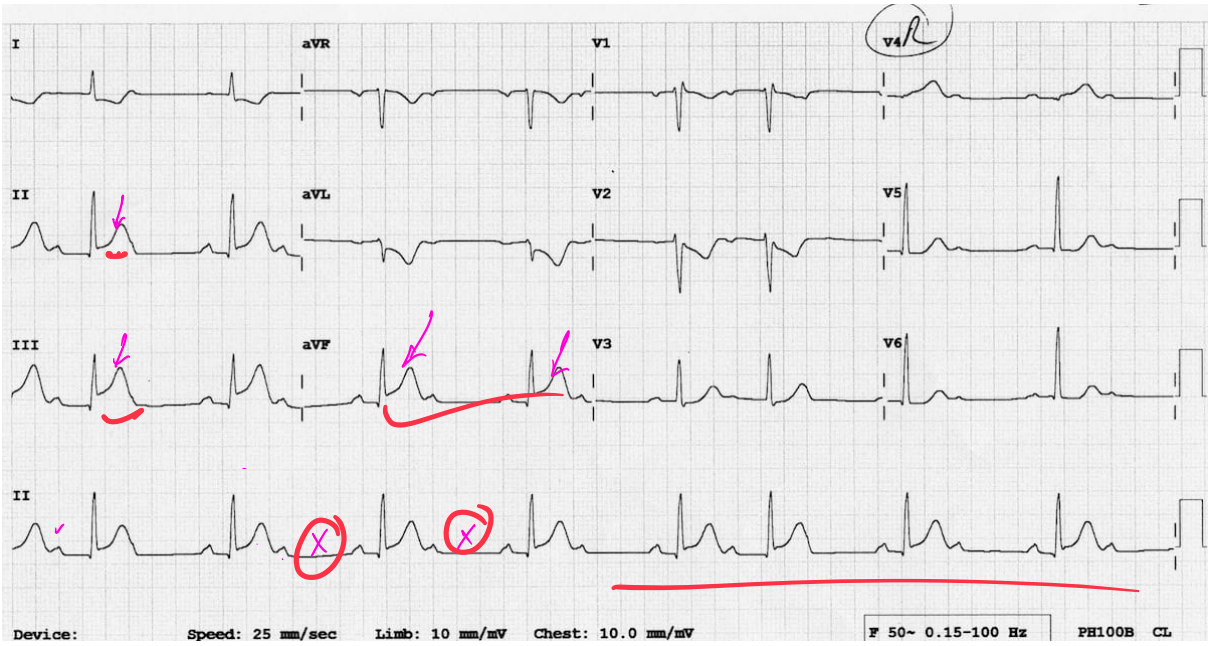

## Mobitz I AV block

Progressive prolongation of PR interval, with a subsequent non-conducted P wave
Repeating 5:4 conduction ratio of P waves to QRS complexes
Relatively constant P-P interval despite irregularity of QRS complexes
**The first clue to the presence of Mobitz I AV block on this ECG is the way the QRS complexes cluster into groups, separated by short pauses**. This phenomenon usually represents 2nd-degree AV block or non-conducted PACs; occasionally SA exit block.



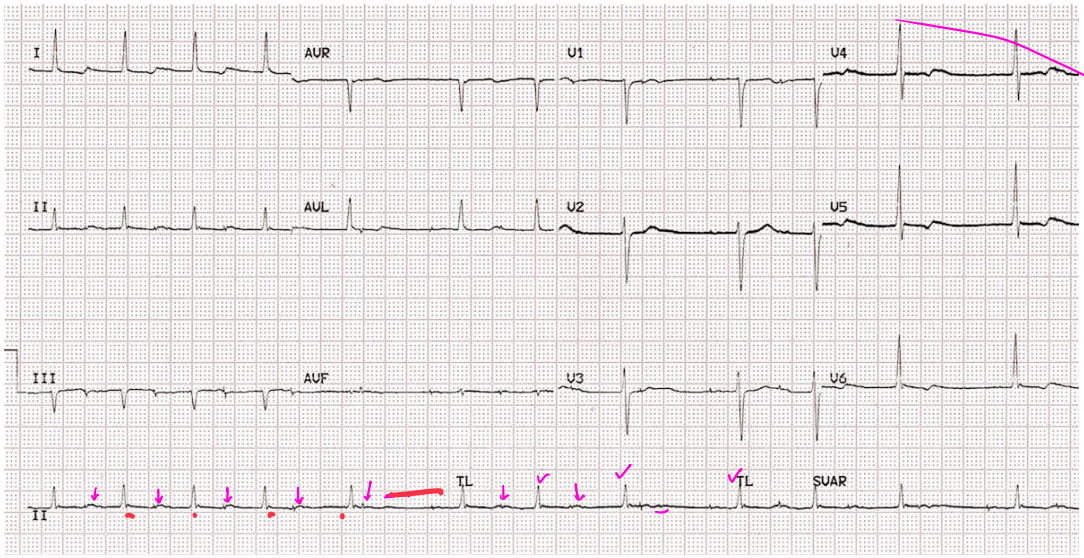

## Mobitz I AV block

QRS complexes clustered in groups, separated by non-conducted P waves.
The P:QRS conduction ratio varies from 5:4 to 6:5.
Note the difference in PR interval between the first and last QRS complex of each group.



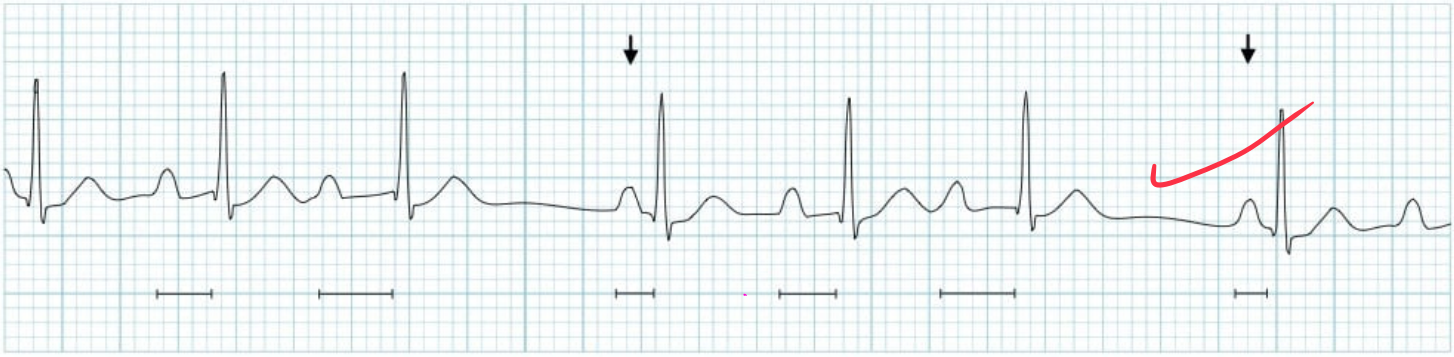

**Mobitz I AV block associated with inferior STEMI and RV infarction**

The majority of the rhythm strip shows 2:1 AV conduction, which makes discerning the type of block difficult (i.e. it could represent Mobitz I or Mobitz II)
However, there is a single 3:2 Mobitz I cycle visible in the middle of the rhythm strip (QRS complexes 5 + 6). If you look hard, you can see a non-conducted P wave deforming the downslope of the T wave in complex 6
Continuous rhythm strip recording revealed that this patient was indeed in Mobitz I AV block

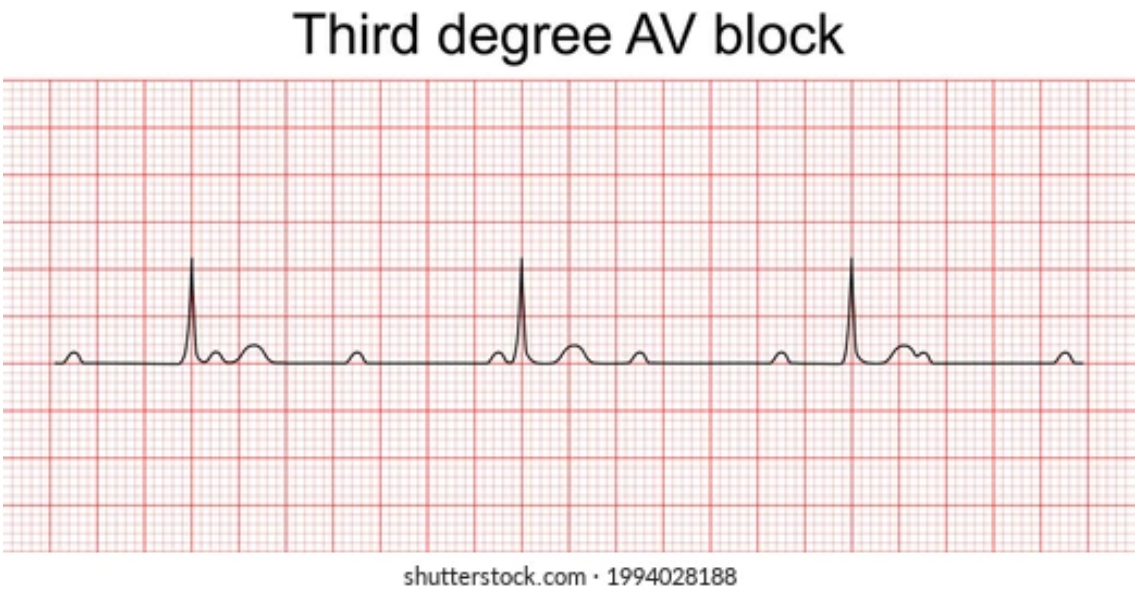



**Mobitz I in a patient with atrial pacing following mitral valve surgery**
**Small atrial pacing spikes precede the QRS complexes.**
The interval between the pacing spikes increases progressively until there is a non-conducted pacing spike.
To find out the story behind this ECG, check out this chapter from the ECG Exigency series:"Post-op Pacing Puzzler"

**This ECG rhythm strip was originally featured on this page as an example of Wenckebach AV block. Can you spot the "deliberate" mistake?**

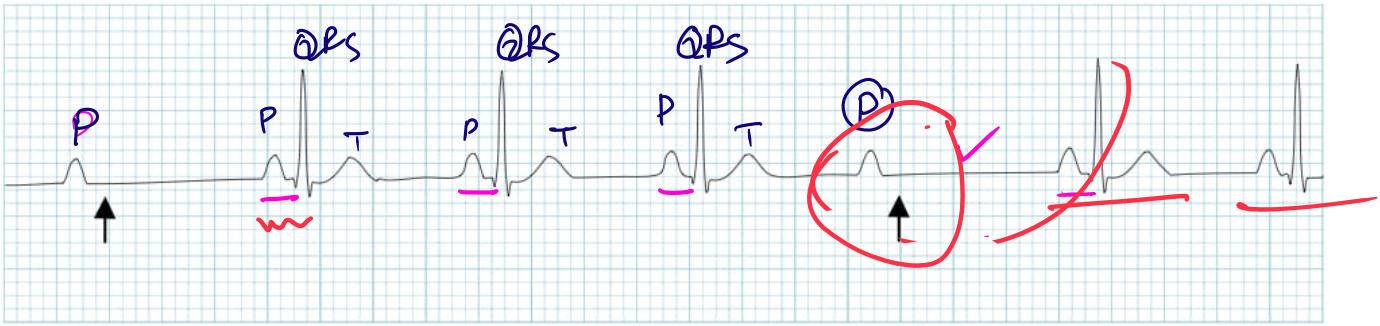


**Q1. What features of Wenckebach AV block are present on this ECG?**
**Q2. What features of Wenckebach are notably ABSENT?**
**Q3. What possible explanations could exist to explain this tracing?**



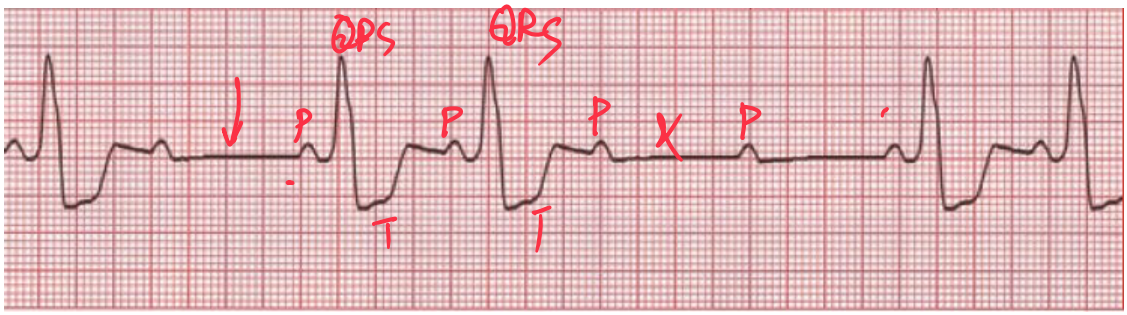
# AV Block: 2nd degree, Mobitz II (Hay block)

A form of 2nd degree AV block in which there is **intermittent non-conducted P waves without progressive prolongation of the PR interval**




Arrows indicate "dropped" QRS complexes (i.e. non-conducted P waves)

The PR interval in the conducted beats remains constant
The P waves 'march through' at a constant rate
The RR interval surrounding the dropped beat(s) is an exact multiple of the preceding RR interval (e.g. double the preceding RR interval for a single dropped beat, triple for two dropped beats, etc)



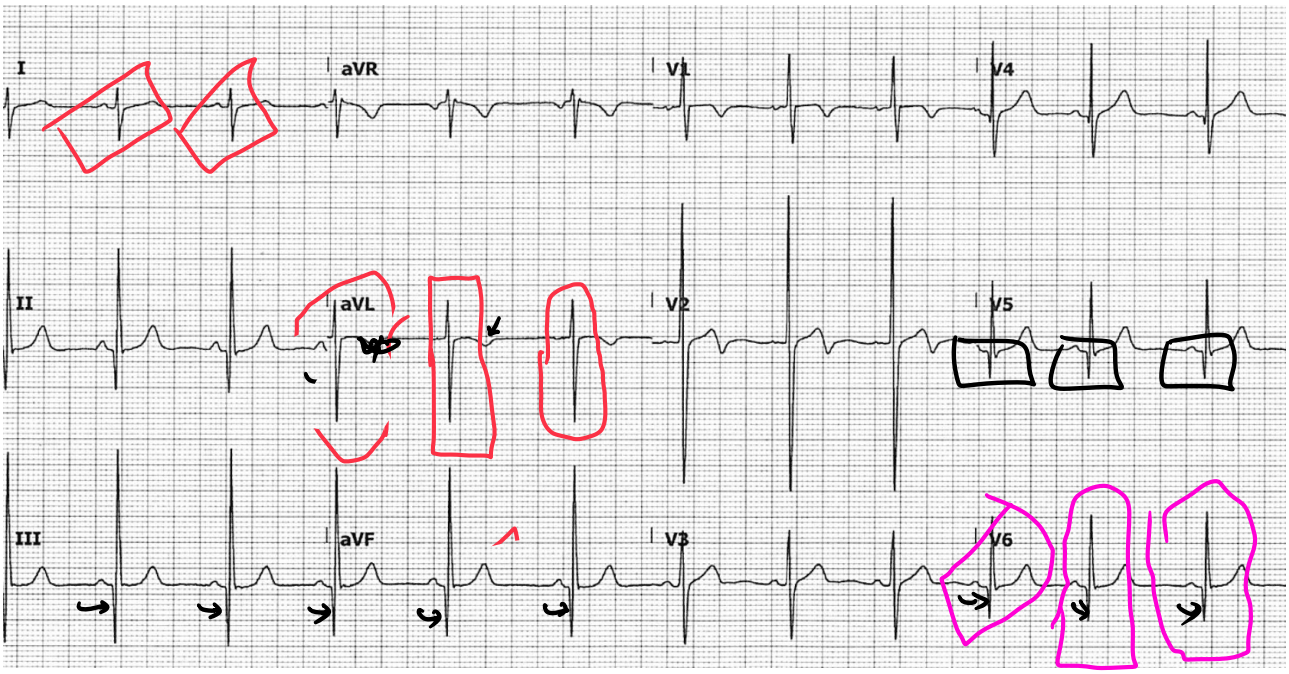

**HOCM**

**Left ventricular hypertrophy** with increased precordial voltages and non-specific ST segment and T-wave abnormalities
Deep, narrow ("dagger-like") Q waves in lateral (I, aVL, V5-6) +/- inferior (II, III, aVF) leads

**Left atrial enlargement ("P mitrale")** — left ventricular diastolic dysfunction may lead to compensatory left atrial hyertrophy

**Signs of WPW (short PR, delta wave)** — ECG features of Wolff-Parkinson-White (WPW) were seen in 33% of patients with HCM in one study,

**Giant precordial T-wave inversions in apical HCM**




**Classical (asymmetrical) Hypertrophic Cardiomyopathy:**

1) Voltage criteria for LVH in precordial and limb leads
2) Narrow, "**dagger-like**" Q waves in **inferior and lateral leads**

**lateral (I, aVL, V5-6) +/- inferior (II, III, aVF)**

## Q wave morphology

Q waves seen in HCM can **mimic prior myocardial infarction, although the Q-wave morphology is different:**

**Infarction Q waves are typically > 40 ms duration**

**Septal Q waves in HCM are < 40 ms**

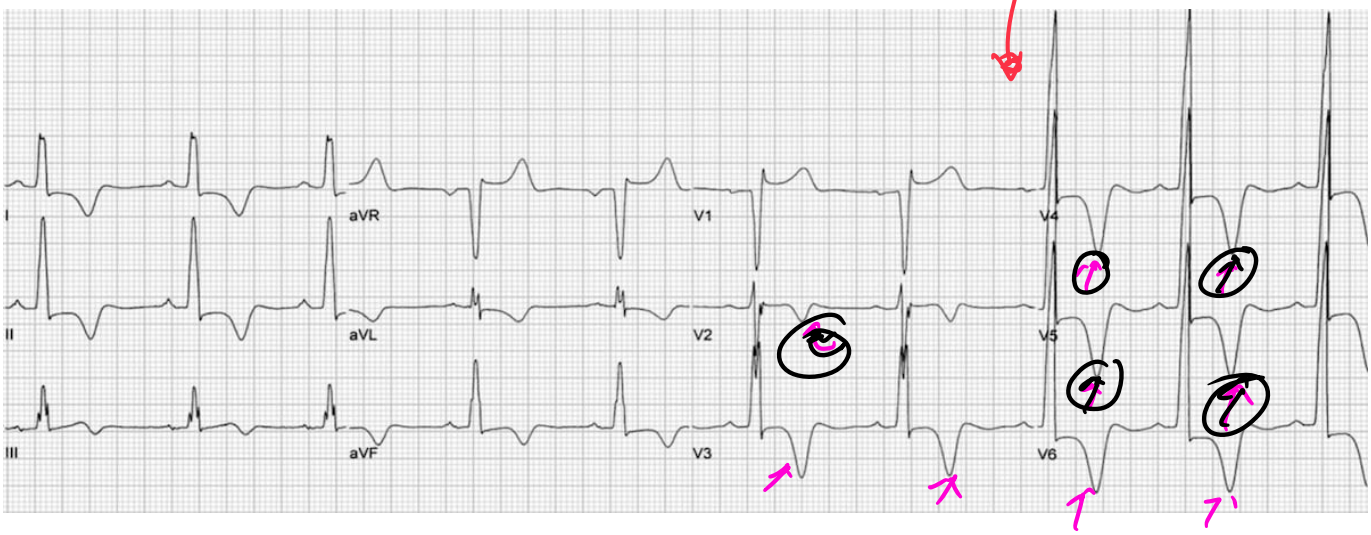
**Lateral Q waves are more common than inferior Q waves in HCM**

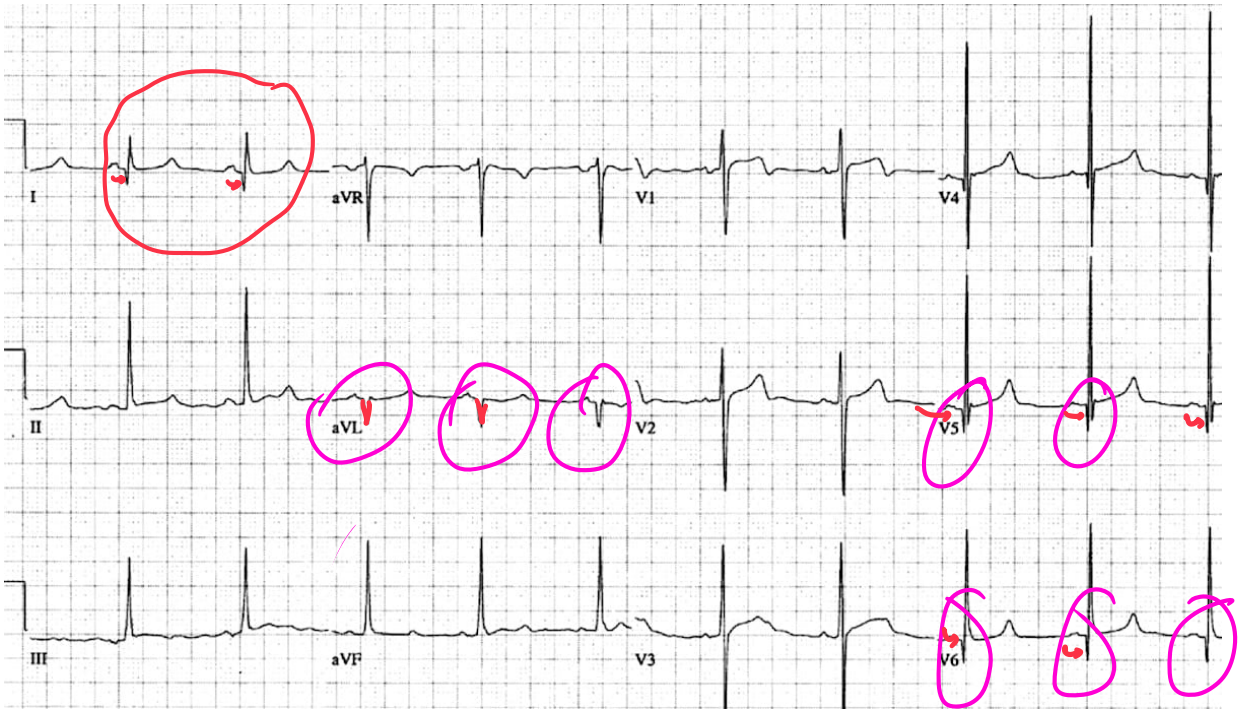
## Apical HCM

This relatively **uncommon form of HCM is seen most frequently in Japanese patients** (13-25% of all HCM cases in Japan)

There is localised hypertrophy of LV apex, causing a "spade-shaped" configuration of the LV cavity on ventriculography

The classic ECG finding in apical HCM is **giant T-wave inversion in the precordial leads**



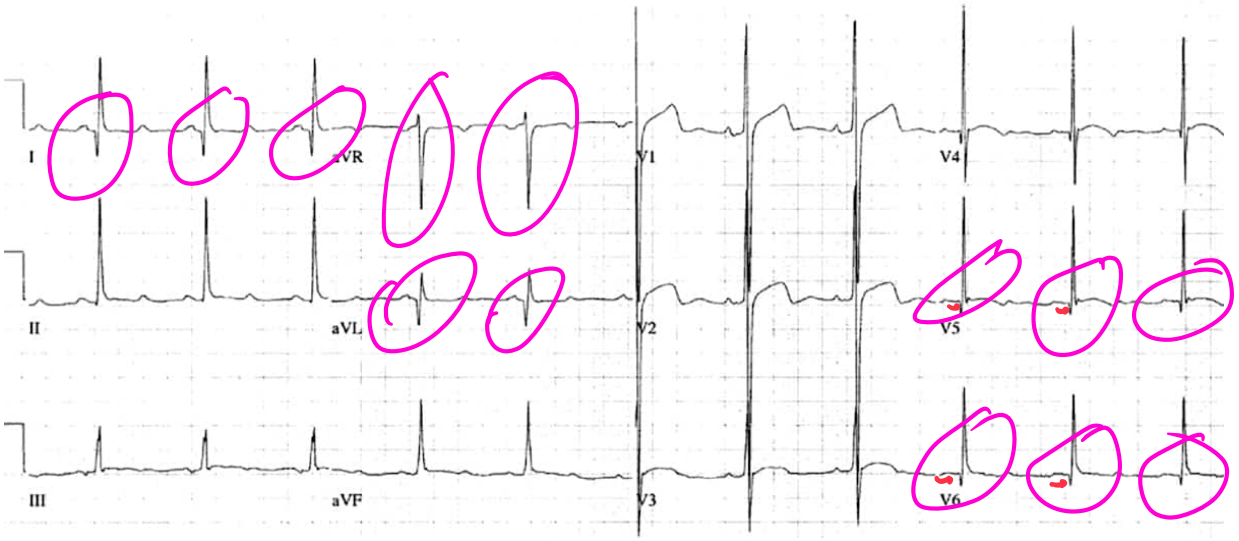



**Deep narrow Q waves < 40 ms wide in the lateral leads I, aVL and V5-6.**

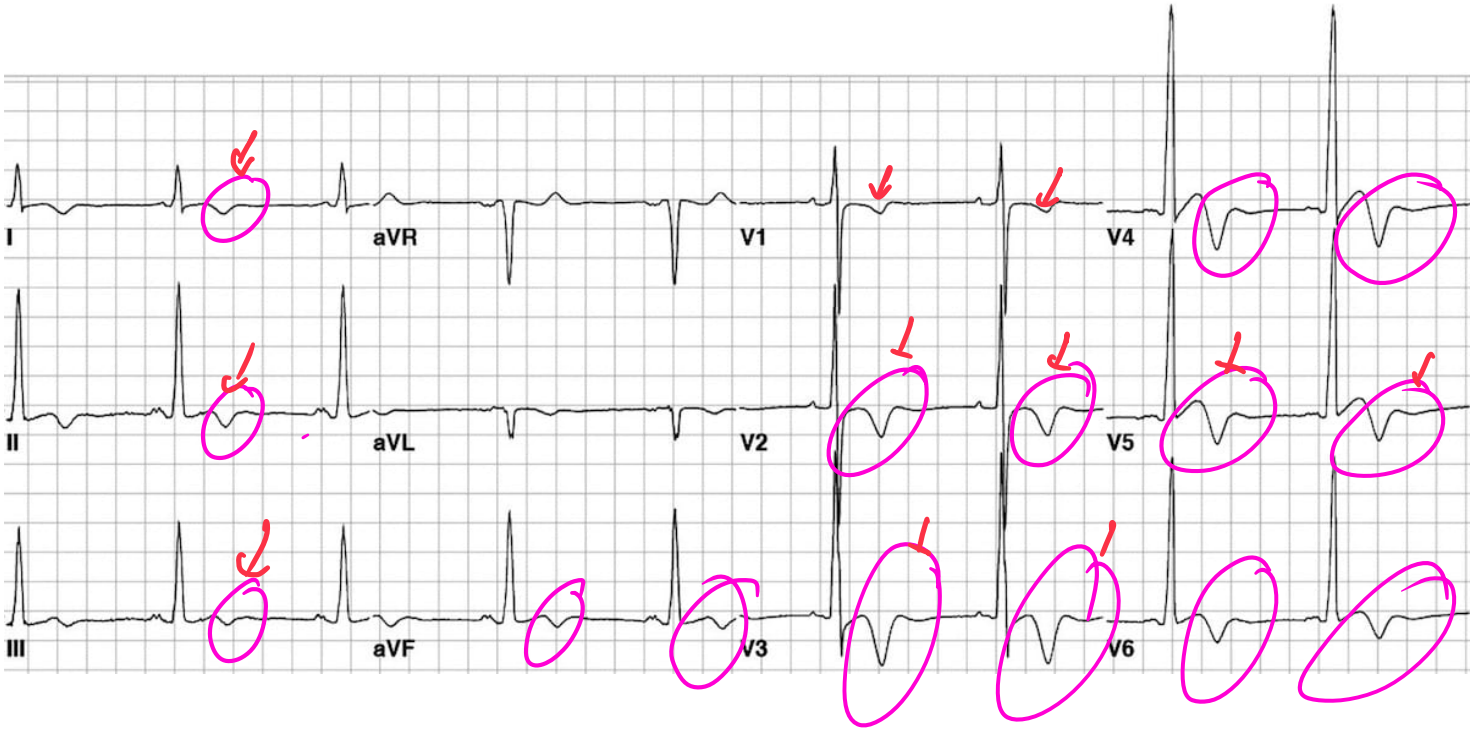
**This ECG was taken from a 30-year old man who presented with exertional lightheadedness and palpitations.** The ECG was misread by the cardiology team as showing "left ventricular hypertrophy, lateral infarct age undetermined".

The patient was discharged home and subsequently **died of a VF arrest** while running to catch a bus. Autopsy showed septal hypertrophy consistent with HCM.

Teaching Point: In a **young patient presenting with exertional symptoms and an ECG that looks this this, think HCM — not "prior lateral infarction"!**

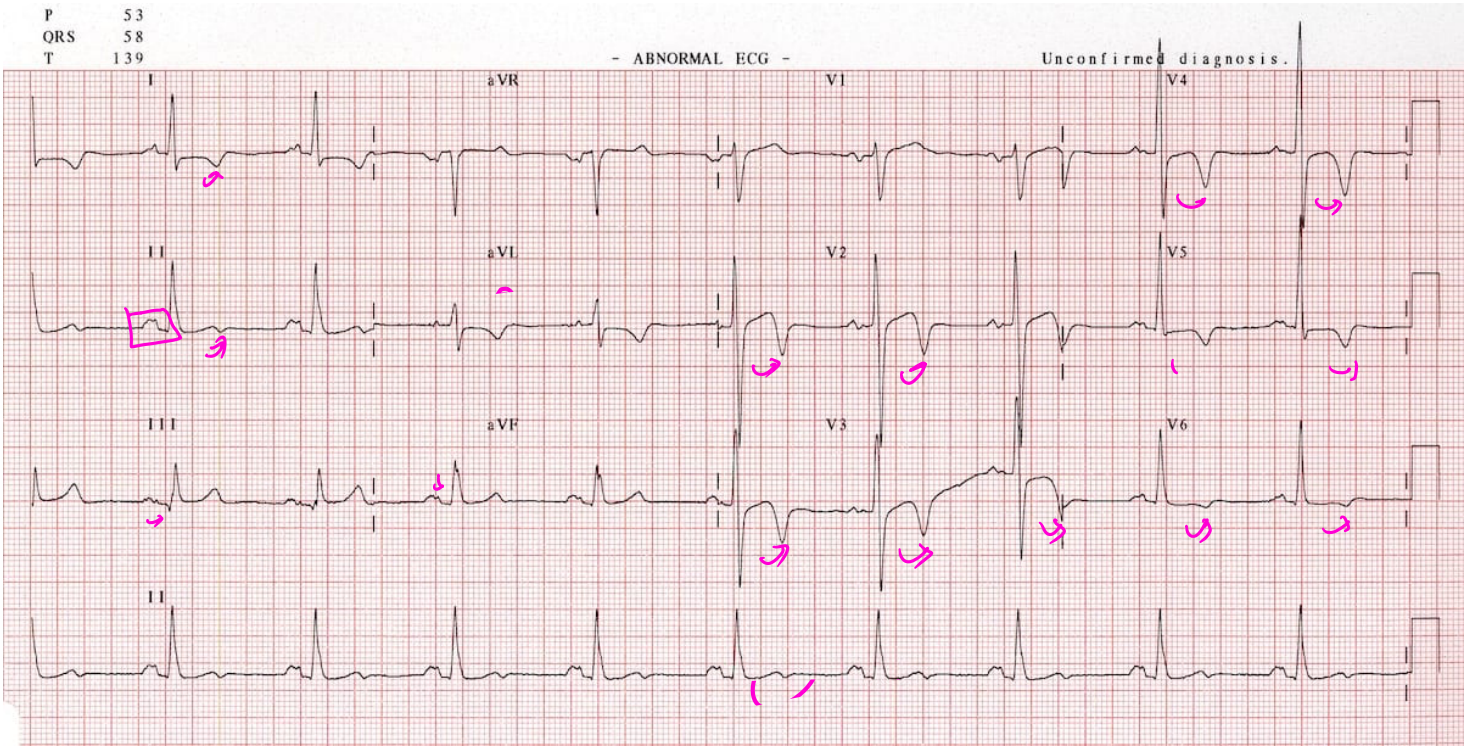



**HCM showing features of asymmetrical septal hypertrophy:**
Large precordial voltages.
Deep, narrow, septal Q waves most prominent in **leads I and aVL; also seen in V5-6.**




**This ECG shows the typical pattern of apical HCM:**

Large precordial voltages.
**Giant T wave inversions** in the precordial leads
Inverted T waves are also seen in the inferior and lateral leads.




**Another example of apical HCM**

High precordial voltages.
**Deep T wave inversions in the precordial and high-lateral leads.**
There is also evidence of **left atrial enlargement ("P mitrale").**

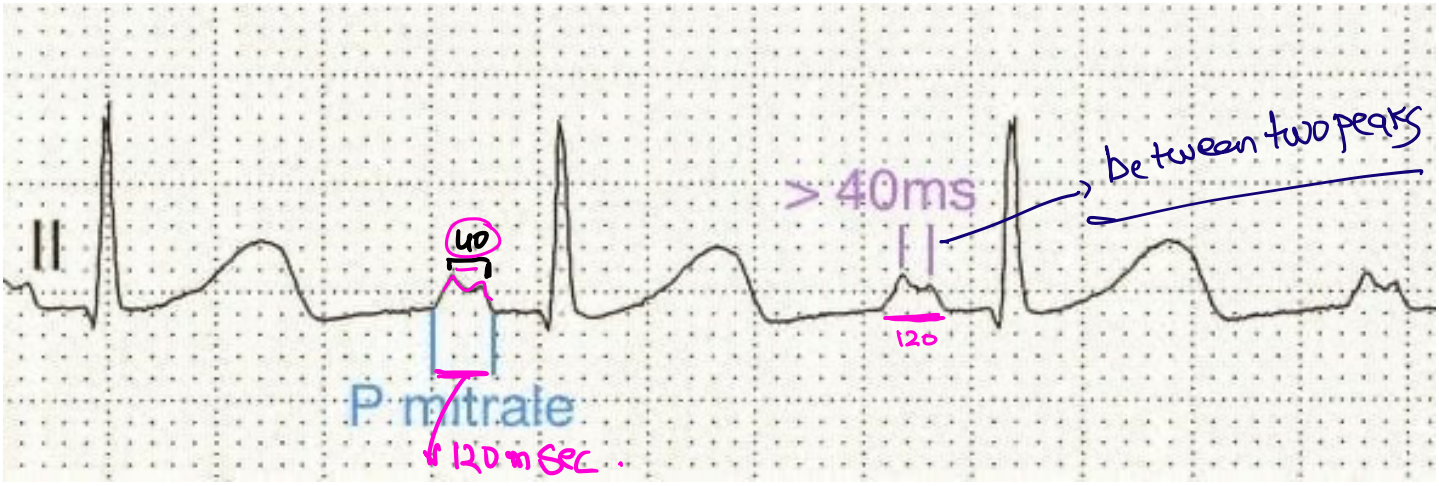
# ECG Criteria for Left Atrial Enlargement

LAE produces a broad, bifid P wave in lead II (P mitrale) and enlarges the terminal negative portion of the P wave in V1.

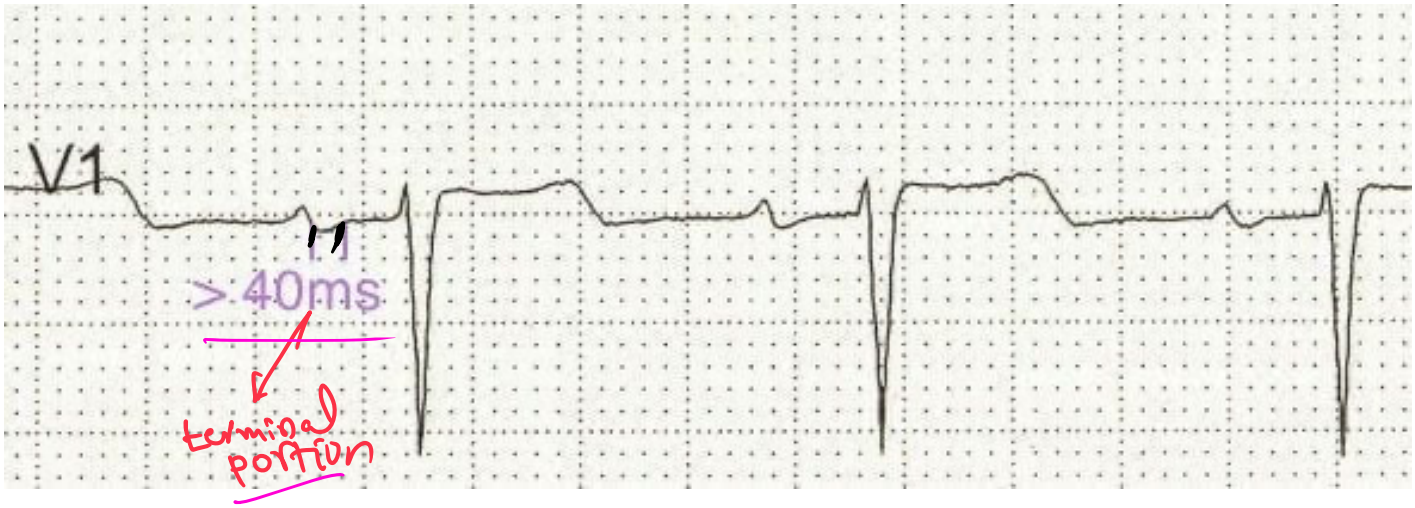
In lead II

Bifid P wave with > 40 ms between the two peaks
Total P wave duration > 110 ms

In V1
Biphasic P wave with terminal negative portion > 40 ms duration
Biphasic P wave with terminal negative portion > 1mm deep

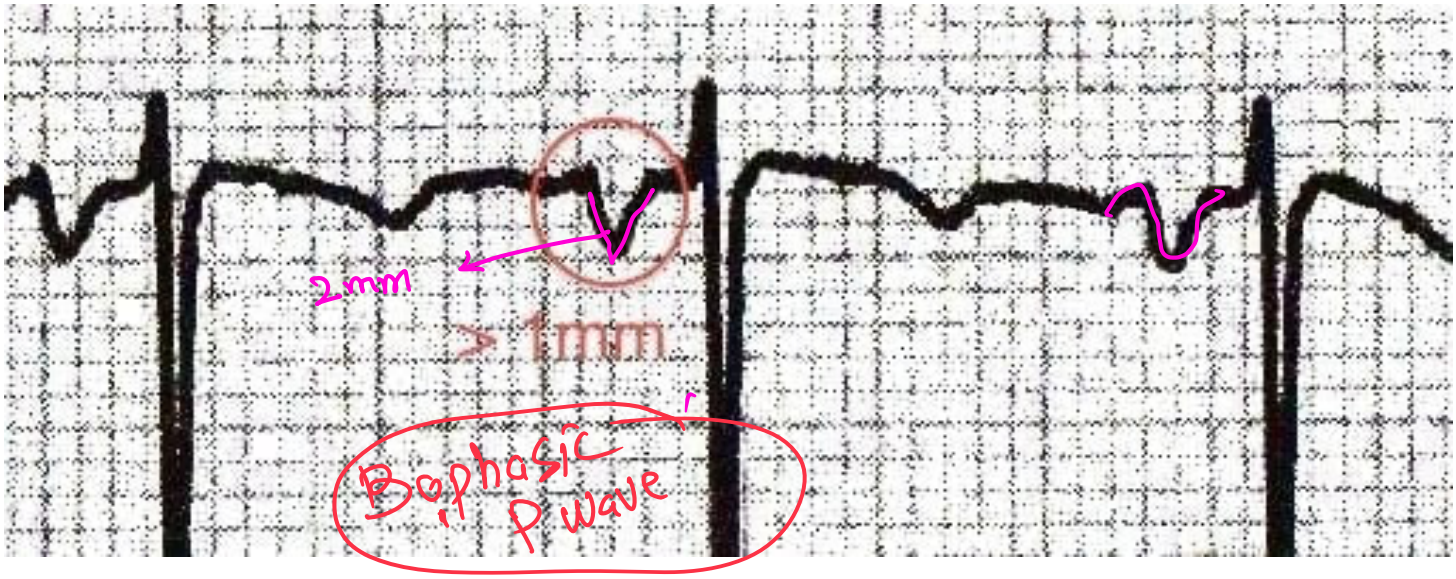



Broad (>110ms), bifid P wave in lead II (P mitrale) with > 40ms between the peaks

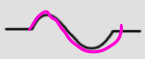



P wave terminal portion > 40 ms duration in V1

Small box (1 mm high) = 0.1mv
Large box (5 mm high) = 0.5 mV




## P waves with terminal portion > 1mm deep in V1

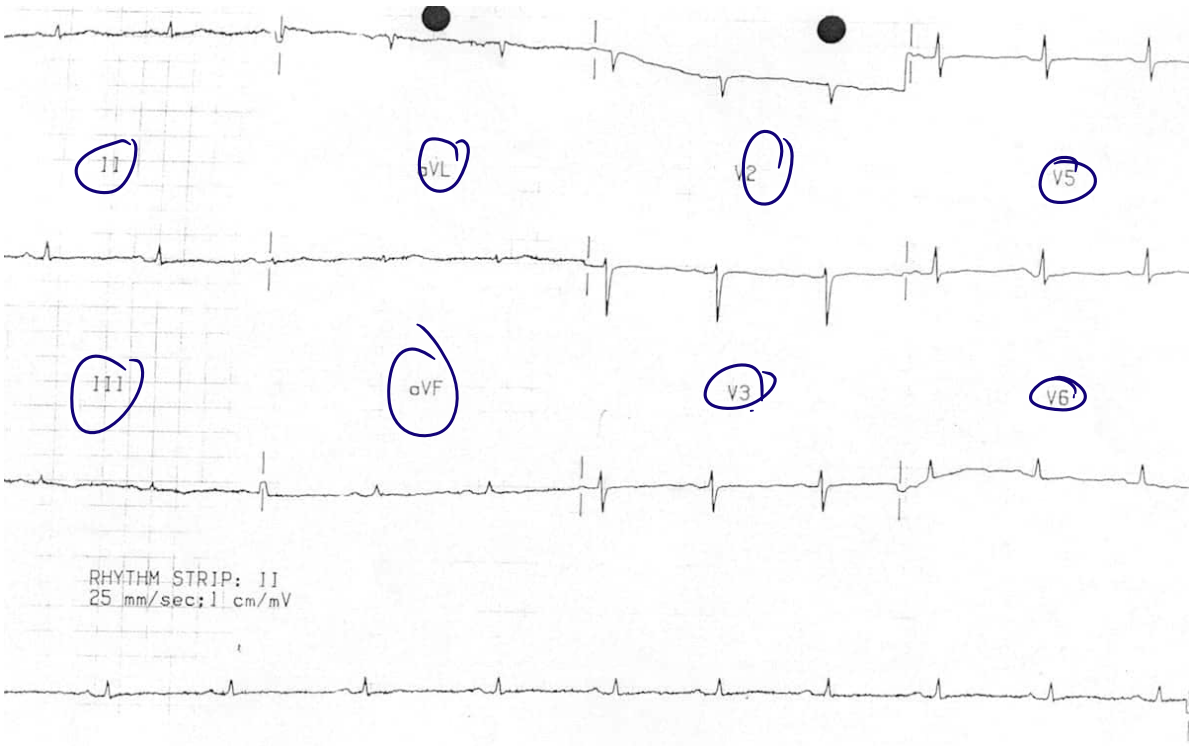
| | II | V1 |
|---|---|---|
| Normal | | |
| RAE | | |
| LAE | |  |
| RAE + LAE | | |

**In II**
**Bifid P wave** with > 40 ms between the two peaks
Total P wave duration > 110 ms

**In V1**
**Biphasic P wave with terminal negative portion > 40 ms duration > 1mm deep**

# ECG Features of Restrictive Cardiomyopathy

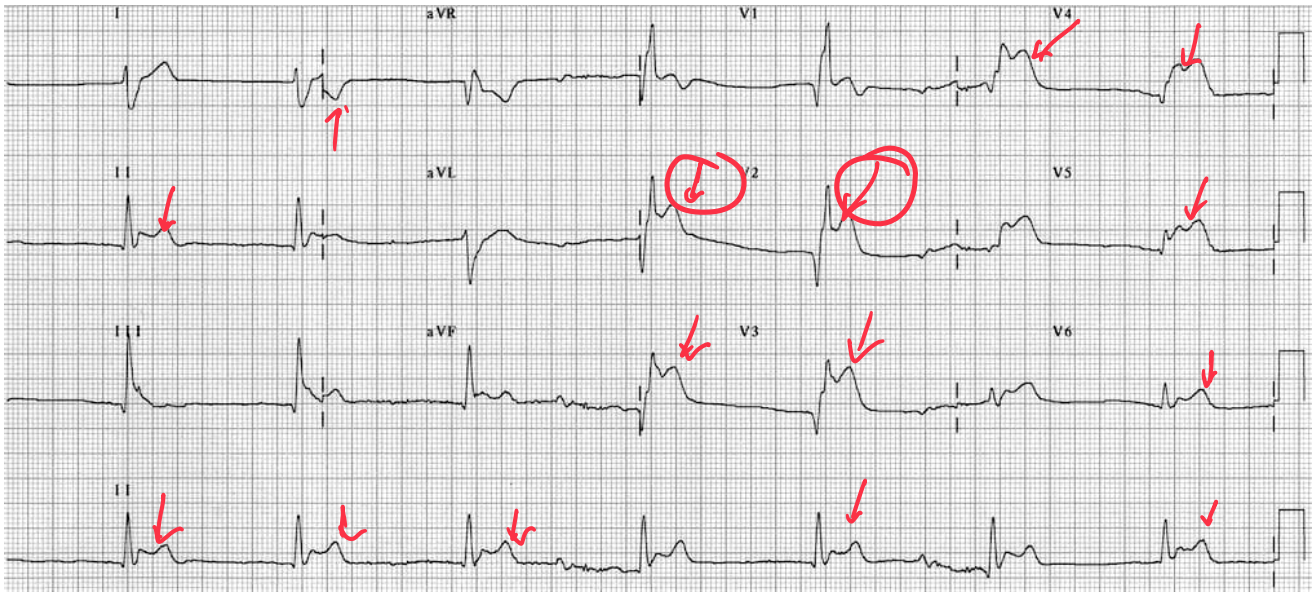
**Low voltage QRS** complexes
Non-specific ST segment / T wave changes
Bundle branch blocks
Atrioventricular block (3rd degree AV block may occur in sarcoidosis)
Pathological "**pseudo-infarction" Q waves**
Atrial and ventricular dysrhythmias




ECG of a patient with **restrictive cardiomyopathy,:**
Low voltage QRS complexes
**Widespread flattening of T waves**

## TAKOTSUBO CARDIOMYOPATHY

**Cardiac enzymes are significantly raised**
**Normal vessels in coronary angiogram**




**Takotsubu Cardiomyopathy: ST elevation is difficult to differentiate from STEMI**

# MYOCARDITIS

**The most common ECG abnormalities seen in myocarditis are:**

Sinus tachycardia

Non-specific ST segment and T waves changes

**Other ECG changes are variable, and may include:**

Prolonged QRS

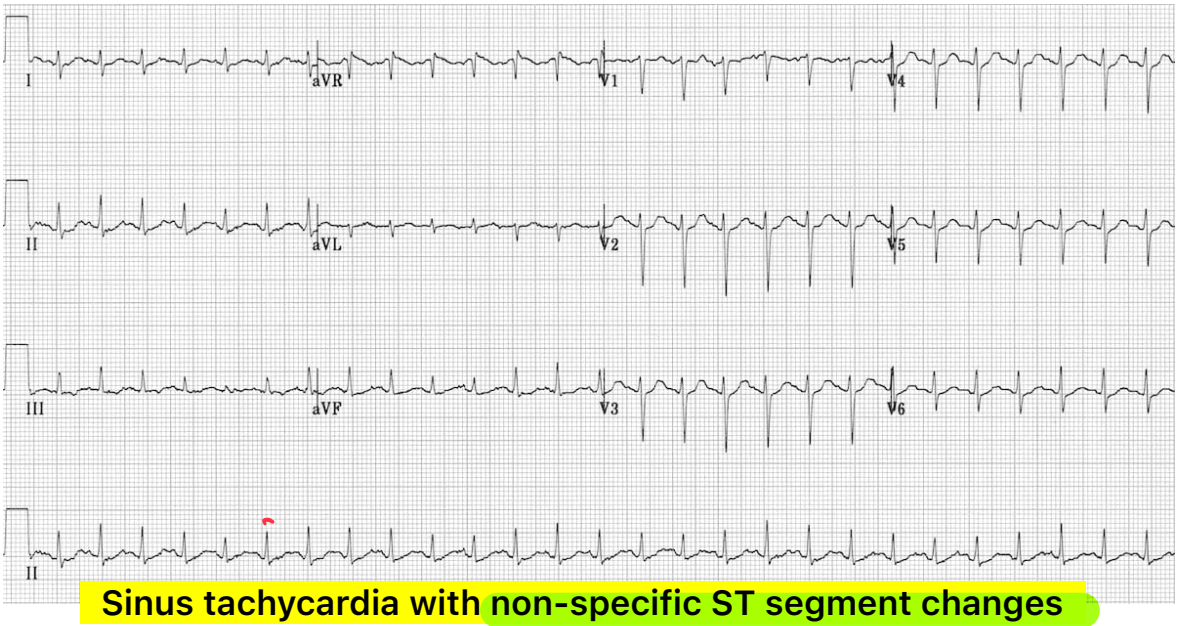
QT prolongation

Diffuse T wave inversion

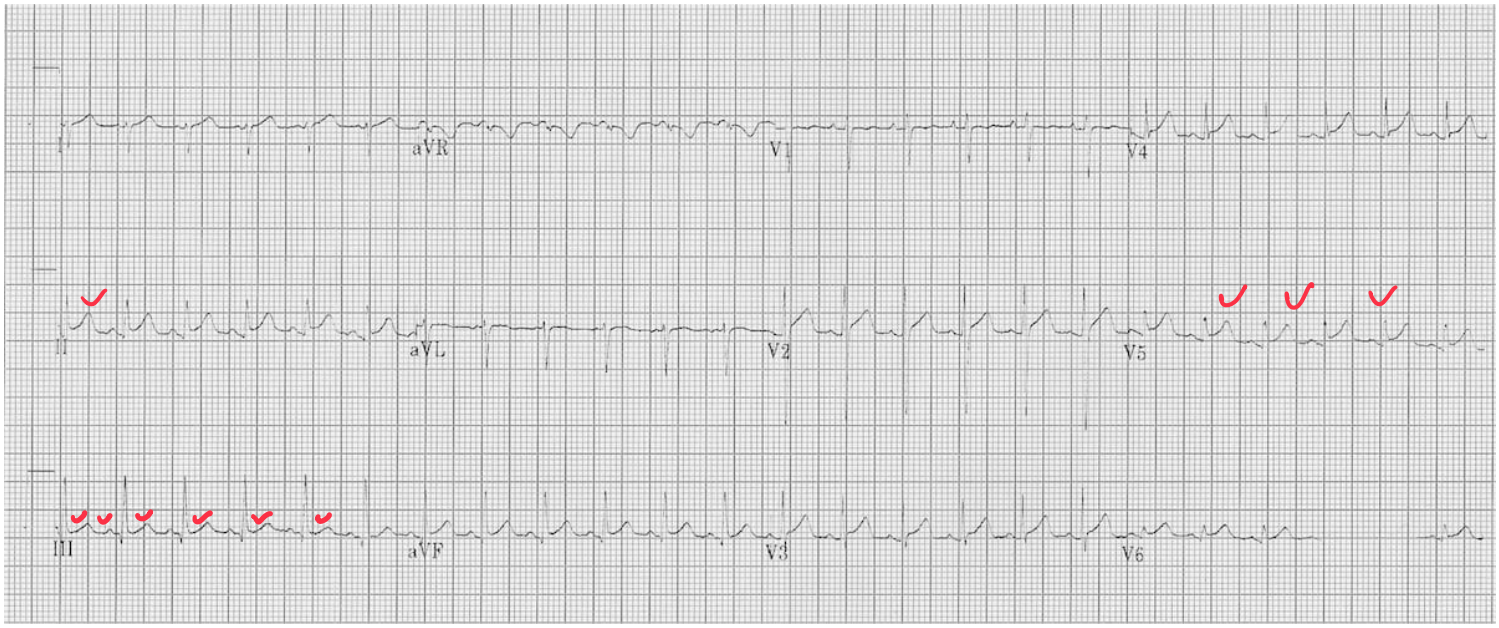
Ventricular arrhythmias

AV conduction defects

With inflammation of the adjacent pericardium, ECG features of pericarditis can also been seen (= myopericarditis)




Sinus tachycardia with non-specific ST segment changes

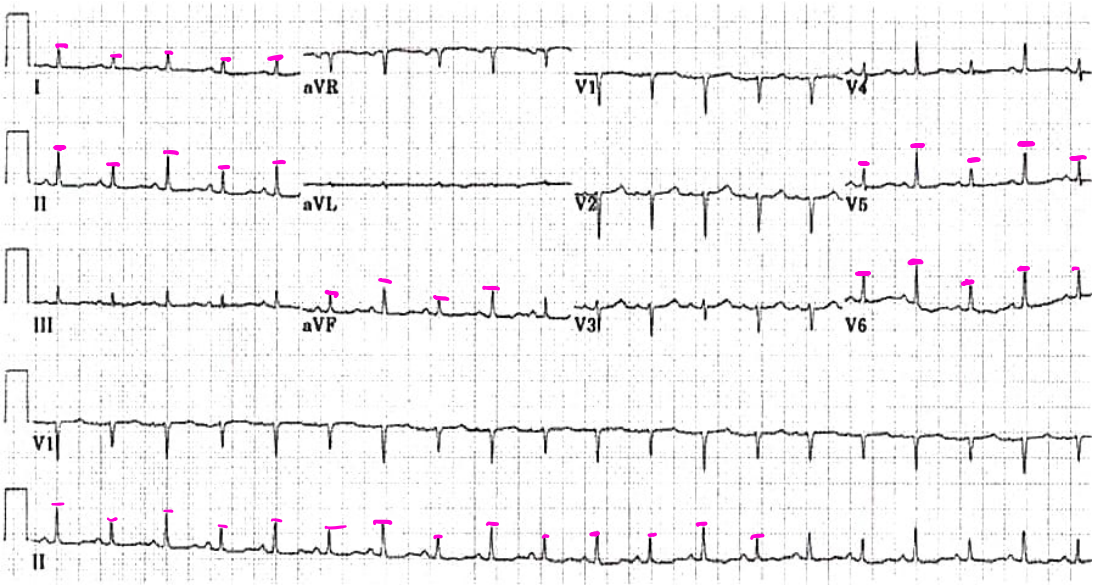



Myopericarditis:

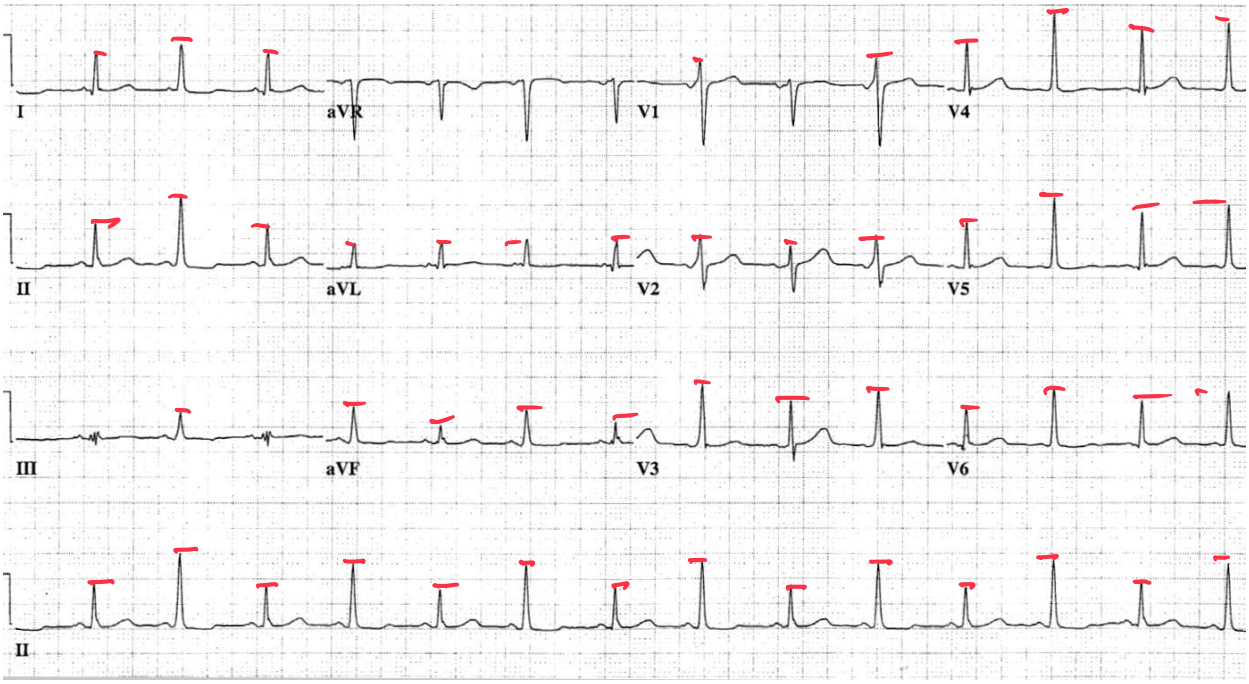
Widespread concave ST elevation (pericarditic changes) in a child with viral myocarditis

# Electrical alternans

Consecutive, **normally-conducted QRS complexes that alternate in height**
Occurs when the heart swings backwards and forwards within a large fluid-filled pericardium

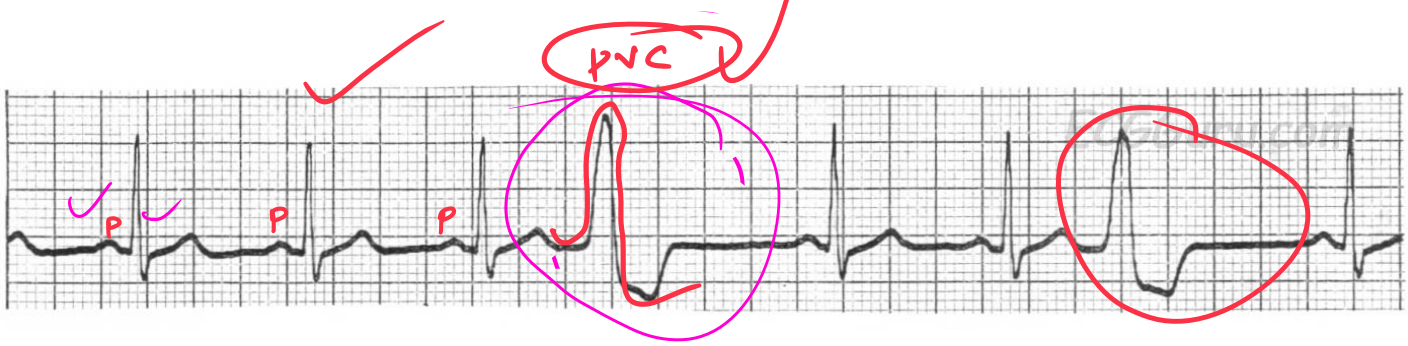



**Electrical alternans: Alternating QRS complex height best seen in lateral precordial leads**




Alternating QRS amplitude and axis.

1) wide
2) fusion
3)




# Left Ventricular Hypertrophy (LVH)

There are numerous voltage criteria for diagnosing LVH, summarised below
The most commonly used are the **Sokolow-Lyon criteria:**
**S wave depth in V1 + tallest R wave height in V5 or V6 > 35 mm**
Voltage criteria **must be accompanied by non-voltage criteria to be considered diagnostic of LVH**

## Voltage Criteria

### Limb Leads

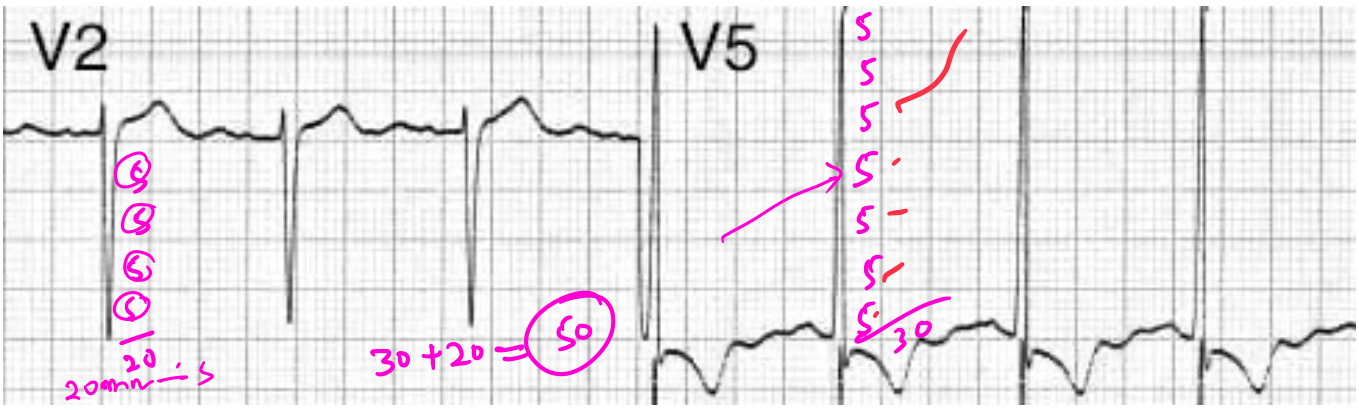
R wave in lead I + S wave in lead III > 25 mm
R wave in aVL > 11 mm
R wave in aVF > 20 mm
S wave in aVR > 14 mm

### Precordial Leads

R wave in V4, V5 or V6 > 26 mm
R wave in V5 or V6 plus S wave in V1 > 35 mm
Largest R wave plus largest S wave in precordial leads > 45 mm

### Non Voltage Criteria

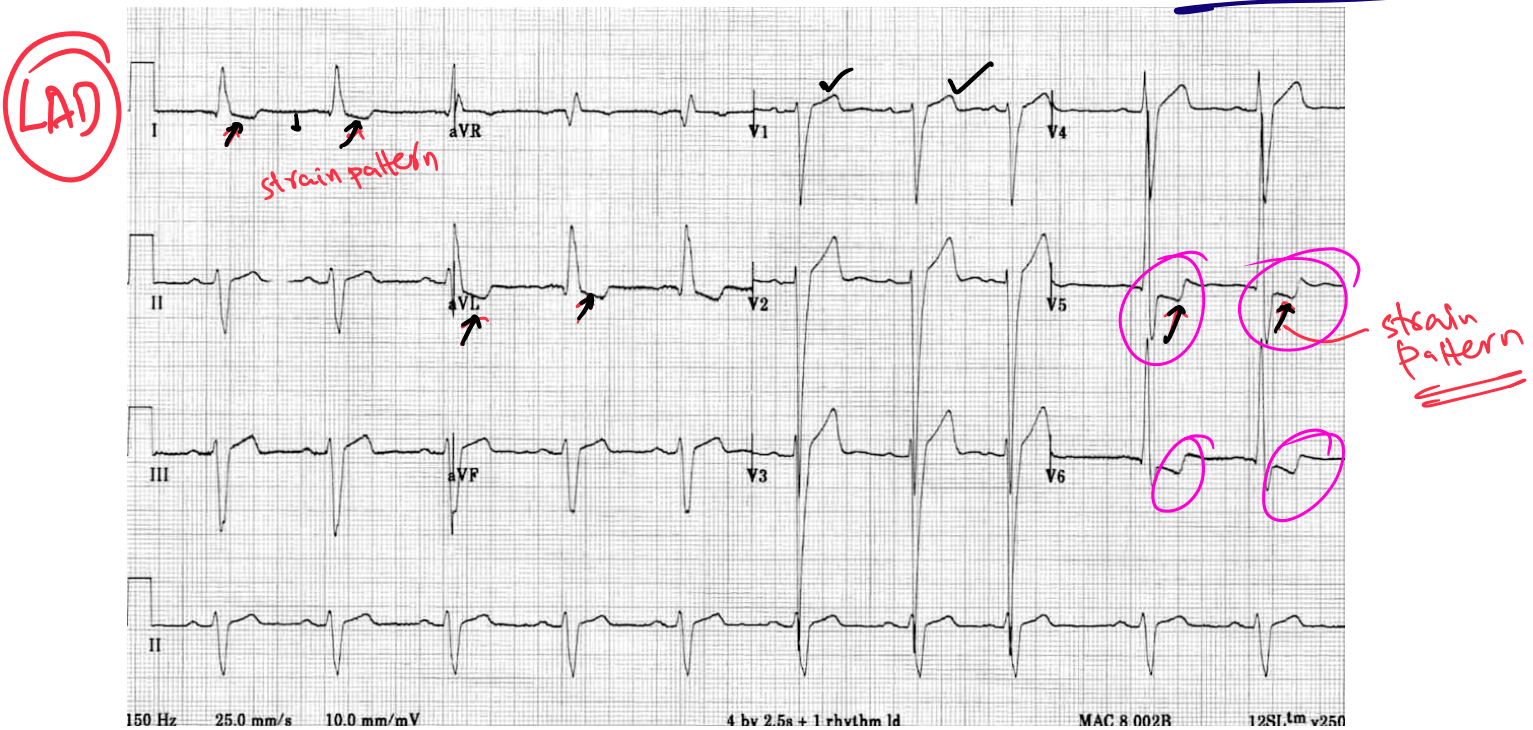
Increased R wave peak time > 50 ms in leads V5 or V6
**ST segment depression and T wave inversion in the left-sided leads: AKA the left ventricular 'strain' pattern**




**LVH by voltage criteria: S wave in V2 + R wave in V5 > 35 mm**

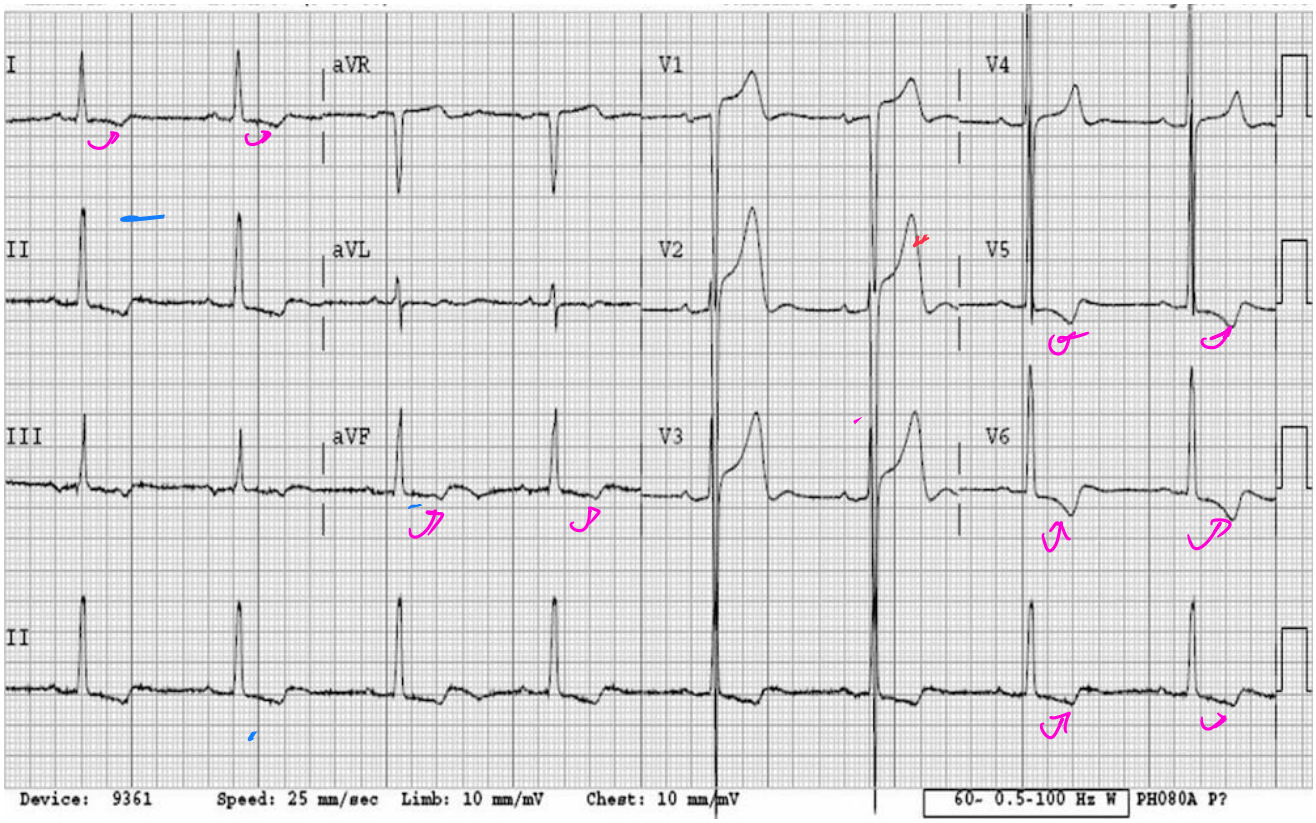
LV strain pattern: ST depression and T wave inversion in the lateral leads

V5 V6 T > aVL




## Left ventricular hypertrophy (LVH):

Markedly increased LV voltages: huge precordial R and S waves that overlap with the adjacent leads (SV2 + RV6 >> 35 mm).
R-wave peak time > 50 ms in V5-6 with associated QRS broadening.
LV strain pattern with ST depression and T-wave inversions in I, aVL and V5-6.
ST elevation in V1-3.
Prominent U waves in V1-3.
Left axis deviation.




There are massively increased QRS voltages — the S waves in V3 are so deep they are literally falling off the page!

The ST elevation in V1-3 is simply in proportion to the very deep S waves ("appropriate discordance").

The LV strain pattern is seen in all leads with a positive R wave (V5-6, I, II, III, aVF).

# Paced ECG – Electrocardiographic Features

## Pacing spikes

**Vertical spikes of short duration, usually 2 ms.**
May be difficult to see in all leads.
Amplitude depends on position and type of lead.
**Bipolar leads result in a much smaller pacing spike** than unipolar leads.
**Epicardially placed leads result in smaller pacing spikes than endocardially placed** leads.

### Atrial Pacing

Pacing spike precedes the p wave.
Morphology of p wave dependent of lead placement but may appear normal.

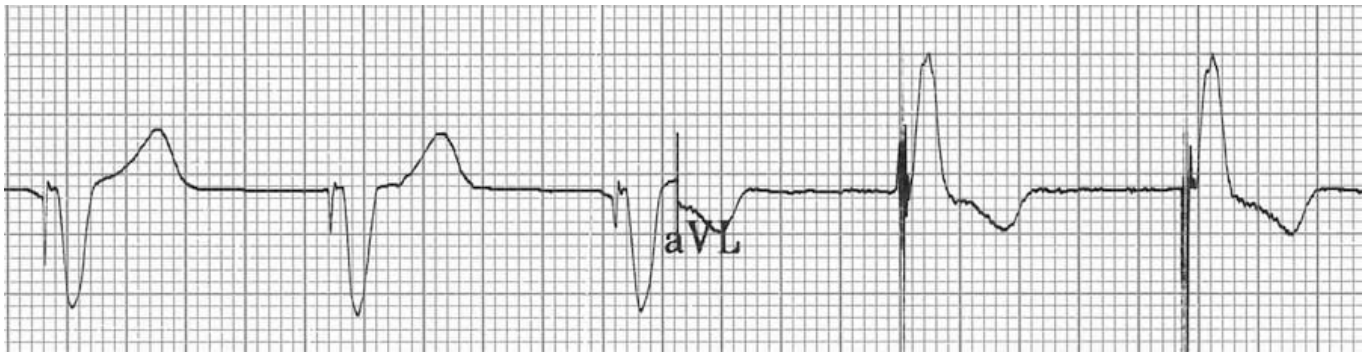
### Ventricular Pacing

**Pacing spike precedes the QRS complex.**
**Right ventricle pacing lead placement** results in a **QRS morphology similar to LBBB**.
**Left epicardial pacing lead placement** results in a QRS morphology **similar to RBBB**.
ST segments and T waves should be discordant with the QRS complex i.e. the major terminal portion of the QRS complex is located on the opposite side of the baseline from the ST segment and T wave.

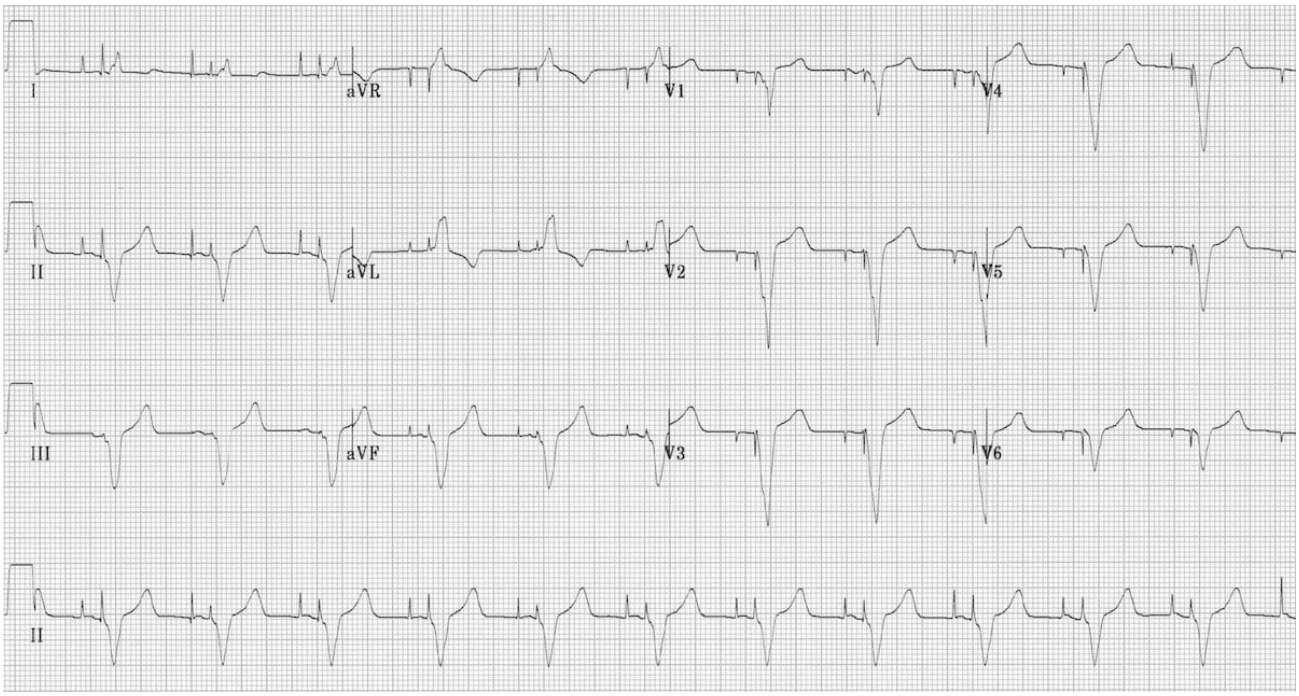
### Dual Chamber Pacing

Dependent on areas begin paced.
May exhibit features of atrial pacing, ventricular pacing or both.
Pacing spikes may precede only p wave, only QRS complex, or both.

Atrial and ventricular pacing spikes

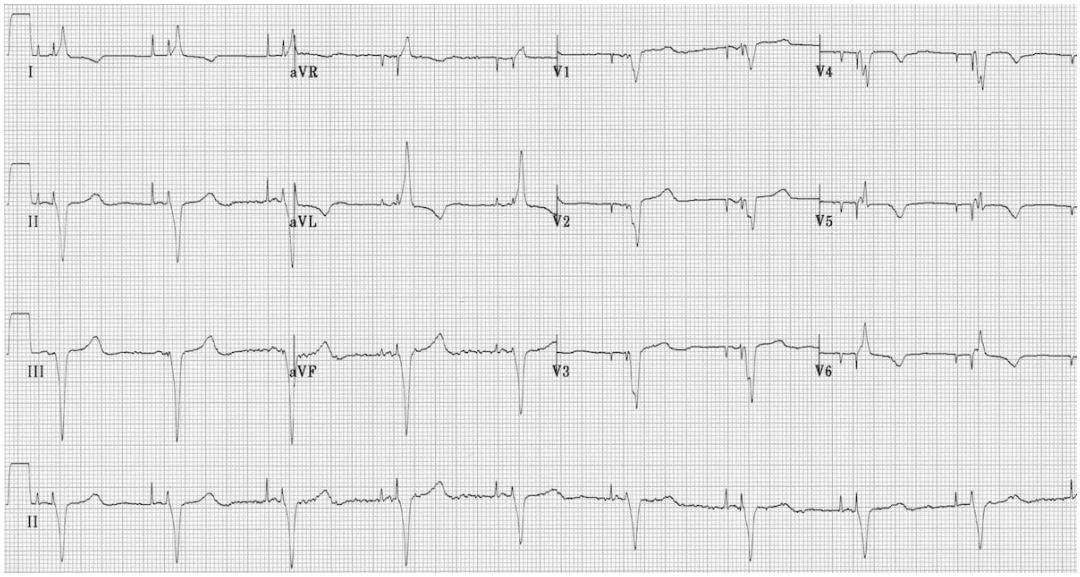



Appropriate discordance in a ventricular paced rhythm



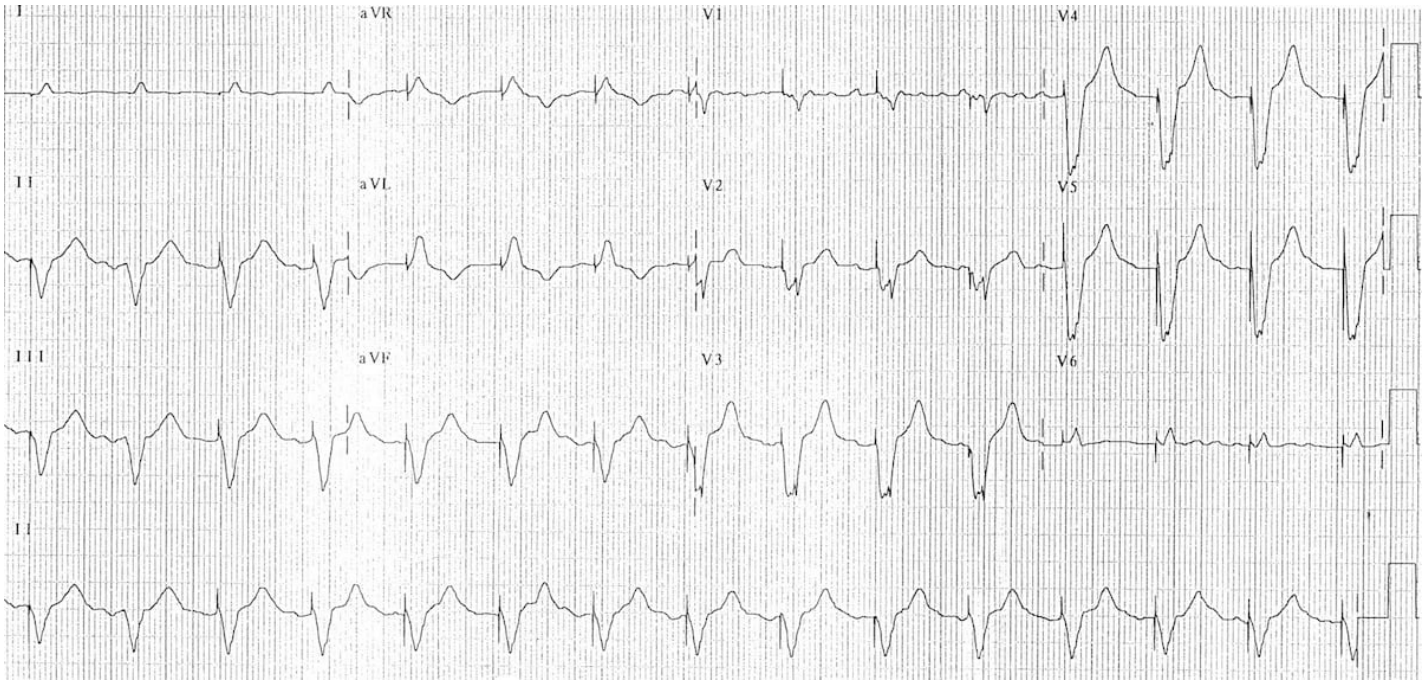

## A-V sequential pacing:

**Atrial and ventricular pacing spikes are visible before each QRS complex.**
There is ***100% atrial capture*** — small P waves are seen following each atrial pacing spike.
There is ***100% ventricular capture*** — a QRS complex follows each ventricular pacing spike.
QRS complexes are broad with a LBBB morphology, indicating the presence of a ventricular pacing electrode in the right ventricle.




**A-V sequential pacing — atrial and ventricular pacing spikes precede each QRS complex with 100% capture.**

## AV SEQUENTIAL



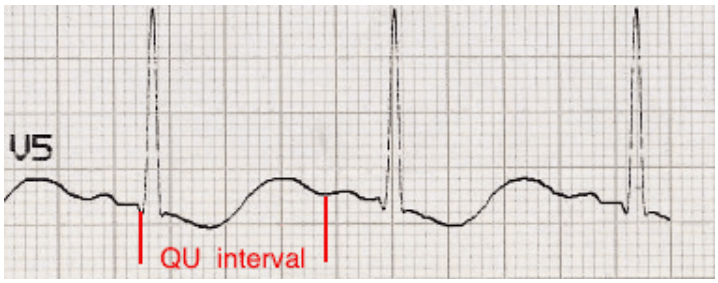

**Ventricular paced rhythm:**

**Ventricular pacing spikes precede each QRS complex** (except perhaps complex #2 — although the QRS morphology in this complex is identical to the rest of the ECG, suggesting that this beat is also paced)
No atrial pacing spikes are seen.
The underlying **native rhythm is probably coarse atrial fibrillation** — there are several possible P waves visible in V1 but otherwise the atrial activity is chaotic.

# Hyypokalemia

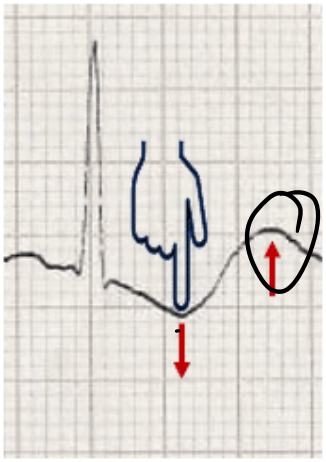
Increased P wave amplitude
**Prolongation of PR interval**
**Widespread ST depression and T wave flattening/inversion**
**Prominent U waves (best seen in the precordial leads V2-V3)**
Apparent **long QT interval due to fusion of T and U waves (= long QU interval)**

**T wave inversion and prominent U waves**



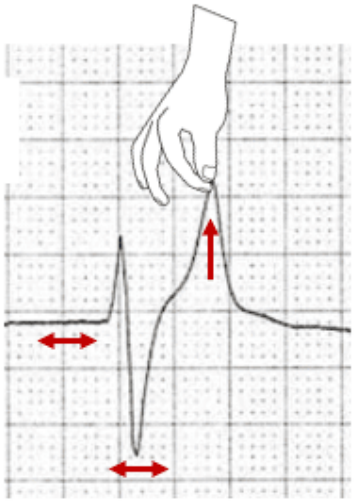

**QU interval: The apparent pseudo-prolonged QT interval is actually the QU interval with an absent T wave**

## The push-pull effect



### Hypokalaemia

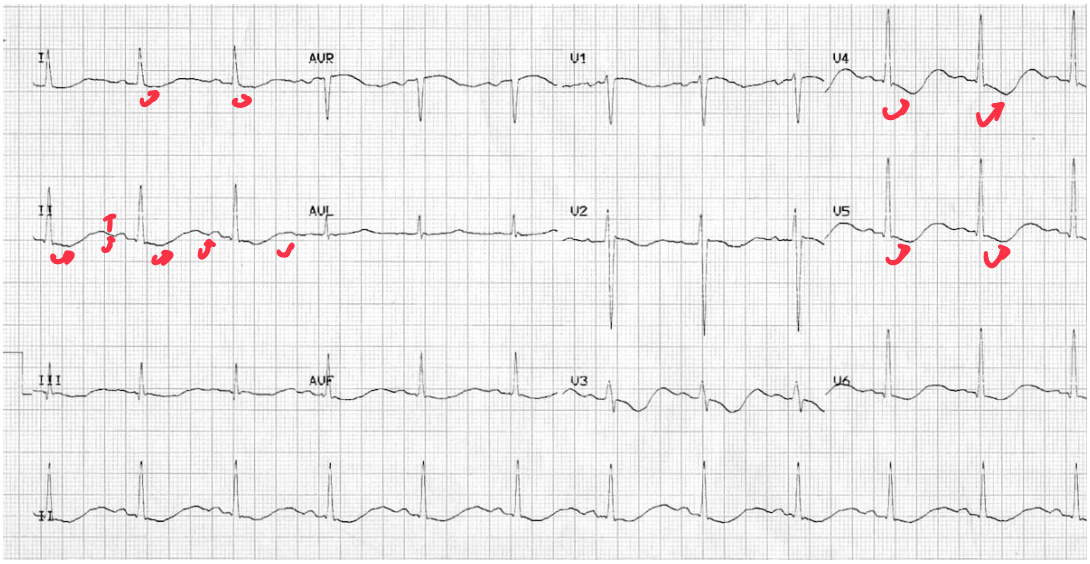
T wave inversion
ST depression
Prominent U wave



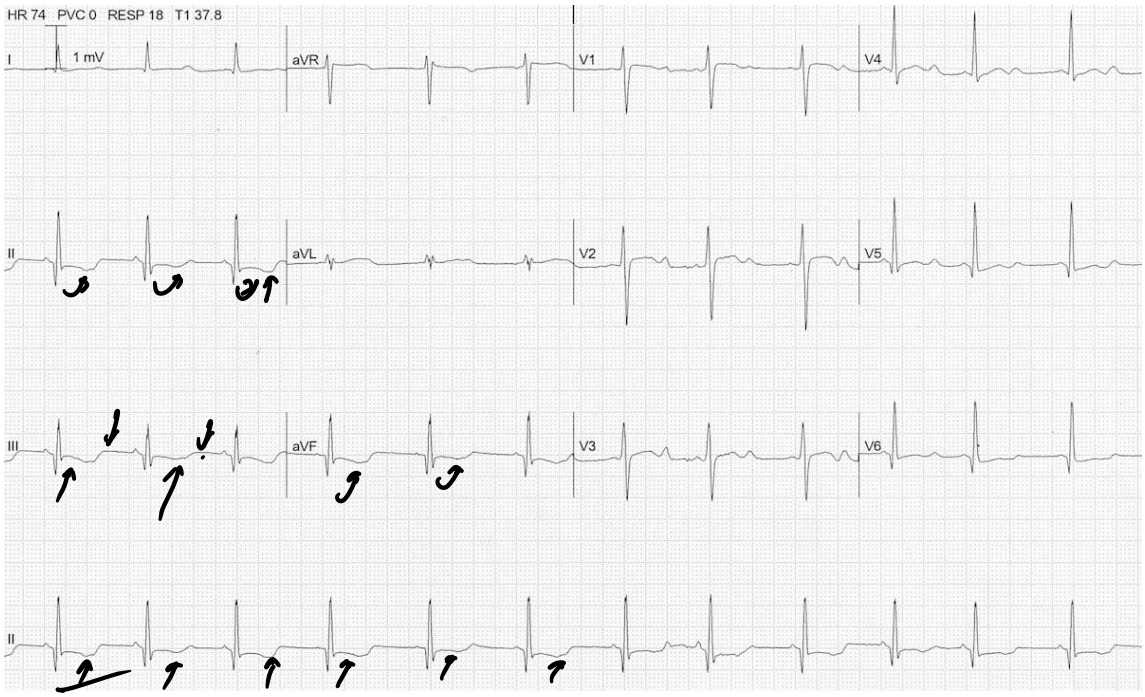
### Hyperkalaemia

Peaked T waves
P wave flattening
PR prolongation
Wide QRS complex

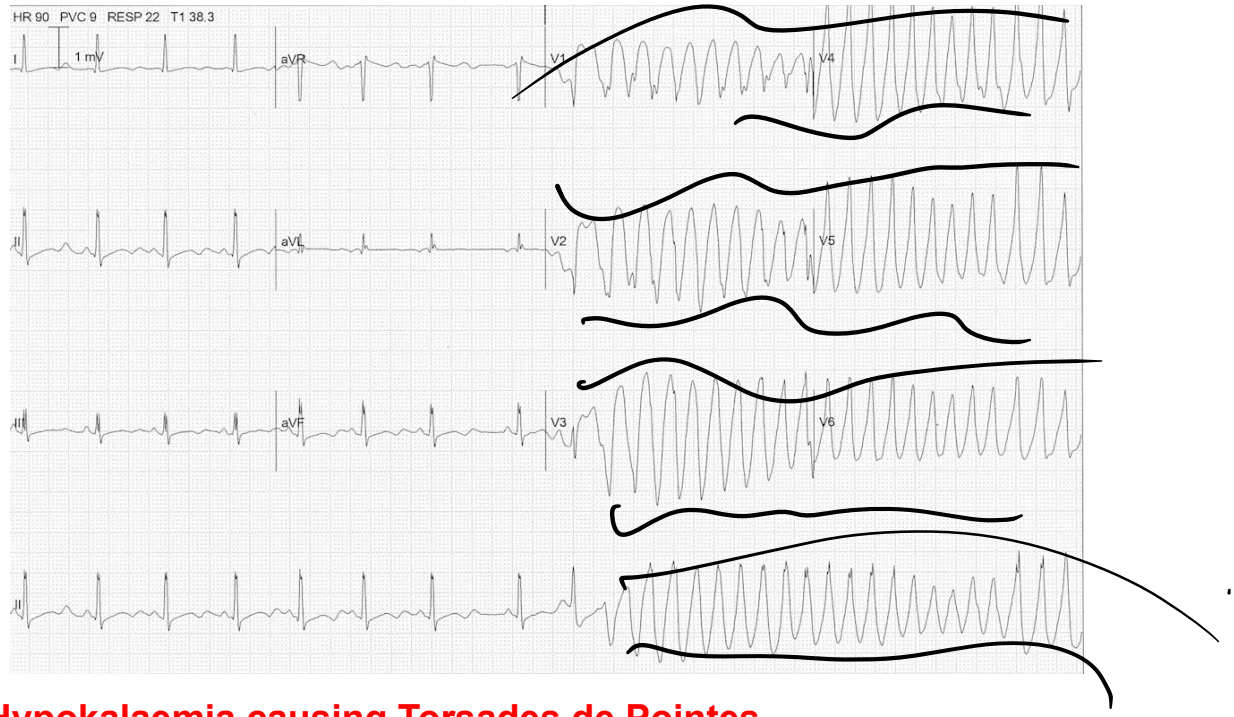
LIFE IN THE FASTLANE




**Hypokalaemia:**
**Widespread ST depression and T wave inversion**
**Prominent U waves**
**Long QU interval**




**ST depression and T wave inversion best noted in inferior leads**
**Prominent U waves**
**Long QU interval**




**Hypokalaemia causing Torsades de Pointes**

**Another ECG from the same patient**
**Note the atrial ectopic causing 'R on T' (or is it 'R on U'?) that initiates the paroxysm of TdP**